

SURGERY OF THE
S H O U L D E R

Surgery of the Shoulder

A F DePALMA, M D

James Edwards Professor of Orthopedic Surgery and Head of the Department, Jefferson Medical College, Philadelphia, Attending Orthopedic Surgeon, Jefferson Medical College Hospital, Philadelphia, Attending Orthopedic Surgeon Methodist Episcopal Hospital Philadelphia, Attending Orthopedic Surgeon, St Agnes Hospital, Philadelphia

With 454 Illustrations



Philadelphia

London

Montreal

J B LIPPINCOTT COMPANY

Surgery of the Shoulder

A F DePALMA, M D

James Edwards Professor of Orthopedic Surgery and Head of the Department, Jefferson Medical College, Philadelphia, Attending Orthopedic Surgeon, Jefferson Medical College Hospital, Philadelphia, Attending Orthopedic Surgeon, Methodist Episcopal Hospital Philadelphia, Attending Orthopedic Surgeon, St Agnes Hospital, Philadelphia

With 454 Illustrations



Philadelphia

London

Montreal

J B LIPPINCOTT COMPANY

COPYRIGHT 1950
BY J B LIPPINCOTT COMPANY

THIS BOOK IS FULLY PROTECTED BY COPYRIGHT
AND WITH THE EXCEPTION OF BRIEF EXTRACTS
FOR REVIEW NO PART OF IT MAY BE REPRODUCED
IN ANY FORM WITHOUT THE WRITTEN PERMIS-
SION OF THE PUBLISHERS

PRINTED IN THE UNITED STATES OF AMERICA

This work is dedicated to my wife, Vivienne, and my sons Bruce, Barton and Brian whose patience and tolerance have made this book possible

Preface

Before any work is contemplated on the shoulder, tribute must be paid to Dr E A Codman whose meticulous and comprehensive investigation recorded in his book, *The Shoulder* has laid down the foundation for all future study in this region. No one has done more to extract from the vague symptom complex "the painful and stiff shoulder" the numerous heterogeneous entities which it comprises. His observations relative to the pathology and the management of lesions of the musculotendinous cuff, subacromial bursa and his concept of the mechanism of dislocation and fracture of the upper end of the humerus have stood up under the critical assessment of many workers and now have been generally accepted. His work has provided the stimulus for much painstaking research which has resulted in valuable contributions to our stock of knowledge on the shoulder. A few of the more recent and outstanding contributions are the work of Inman, Saunders and Abbott on the functional mechanism of the shoulder, the study of McLaughlin and D Bosworth on lesions of the musculotendinous cuff and that of Hitchcock and Bechtol and of Lippmann on lesions of the tendon of the long head of the biceps brachii muscle.

The writing of this book has been prompted by the desire to assemble under one cover the accumulated knowledge on the anatomy and the physiology of the shoulder and some of the more common entities responsible for dysfunction of the shoulder, also to record the observations made referable to the variational anatomy and the degenerative lesions of the inner side of the scapulohumeral joint noted in successive decades. Knowledge of these alterations enables one to formulate the norm for the different age periods and facilitates

comprehension of the many pathologic processes implicating the musculotendinous cuff and the biceps tendon which may result in impairment of function.

An attempt has been made to interpret the clinical significance of the degenerative abnormalities observed in shoulders of individuals in different decenniums. For example, as will be shown in the text, wear and tear and senescence are responsible for pulling away of the labrum glenoidale from its bony attachment on the glenoid brim, this lesion increases in severity in each successive decade. In the light of this information it becomes apparent that the labrum glenoidale is in no way associated with the stability of the glenohumeral joint. The operations designed to restore the normal relationship of the labrum with the hope that a cure for recurrent dislocations of the shoulder will be affected are based on an erroneous premise. On the other hand this same study on degenerative lesions provides an explanation of the success of some and the failure of other procedures.

The observations made on a series of shoulder joints obtained postmortem from individuals on whom an examination of the extremities was made prior to their deaths are of particular clinical importance and therefore should be reported. It was noted that large defects in the cuff are compatible with good function. This information demands that we revise our concept of the size of a rupture of the cuff which may cause impairment of function and our present-day management of these lesions. However, one is well aware that the aforementioned degenerative lesions differ from traumatic rupture sustained suddenly. In the former nature has ample time to rearrange the mechanics of the shoulder joint so that dysfunction does not ensue. In the

latter this readjustment is lacking hence, marked impairment of function results. However if given time, nature in many instances will make the necessary alterations to restore function.

Many readers may believe that the manner in which the material was obtained for the investigation on "Degenerative Lesions Compatible with Good Function" was unorthodox. That the method was such is true nevertheless in no other way can such material be obtained in sufficient quantities to allow adequate evaluation from a clinical viewpoint of the lesions found. One is forced to admit that the significance of the observations justified the method.

The chapter dealing with frozen shoulder and bicipital tenosynovitis emphasizes that although these are two distinct entities they are closely related. Many workers do not agree with this concept however gross and microscopic studies made on many anatomic and postmortem specimens and on subjects at the operating table support this belief. Whether the pathology the pathogenesis and the management recorded of the above lesions is or is not accepted it is hoped that sufficient interest will be aroused to stimulate further investigation which will either support or disprove the interpretation of the observations made in this chapter.

The theory of the pathogenesis of recurrent dislocation of the shoulder is not an original one. Magnuson in describing his operation for recurrent dislocation of the shoulder referred to muscular imbalance about the shoulder as the causative factor responsible for the malady. This observer and many others believe that any method that will limit external rotation of the shoulder will effect a cure. With this concept the author is in complete agreement and makes a plea that the most simple operative procedure which will achieve restriction of external rotation of the extremity be adopted.

It has been necessary to draw heavily from numerous sources in order to compile

all the material deemed essential in this book. Every effort possible has been made to give full credit to the authors of the material used however, it may be possible that some omissions have occurred unknowingly. In such instances, the author begs to offer his apologies and expresses his regret.

It is hoped that the practitioner and the surgeon will find this book a valuable source of information in the more common pathologic conditions affecting the shoulder region. In addition it is hoped that the studies recorded herein will result in more adequate comprehension of the pathogenesis of these lesions, increased acumen in diagnosis and more effective methods of management.

The author makes the following acknowledgments with a deep feeling of gratitude first to Dr. G. A. Bennett, Director of the Department of Anatomy of the Jefferson Medical College who together with Dr. G. Callery and the author, conducted the investigation on Variational Anatomy and Degenerative Lesions of the Shoulder Joint recorded in Part I of Chapter 3. Dr. Bennett was more than a collaborator in this study. He provided the stimulus necessary to guide the work to a successful termination. His profound knowledge of anatomy and its practical application, together with his keen powers of observation made possible the many significant findings noted. The author is deeply indebted to Dr. Bennett for the many hours of detailed instruction in the manner of approach and conduction of a problem in research, also for the use of the anatomic specimens which were employed in subsequent studies in the shoulder and for the advice, guidance and encouragement that he readily gave. All of the illustrations on the glenoid side of the scapulohumeral joint used in this book were obtained from the investigation conducted together with Dr. Bennett and Dr. Callery.

Acknowledgment is also due to Dr. P. C. Swenson, Professor of Radiology, Jefferson Medical College who so kindly permitted us to use all the facilities of his department.

Dr R L Breckenridge, who gave valuable aid in the interpretation of the microscopic sections, Dr J B White who faithfully assisted in the study on the Lesions of the Shoulder Joint Compatible with Good Function, Dr G Diebert, Dr J Gartland and Dr T Armstrong, who helped collect the gross specimens and the case histories used in this work. Dr M Blaker and Dr I Kaplan who gathered much material and prepared the bibliography and the index, Mrs M Gross, who so carefully edited the manuscript my able office staff, Miss T Everett and Miss V Quick for their painstaking

work on the many drafts of the manuscript and the arrangement of the illustrations. Mr A Hancock, the photographer for the Jefferson Hospital, for his conscientious and meticulous work the editor and publishers of the Instructional Course Lectures, for the privilege of using material which appeared previously in this publication, Mr C Brill, the artist, who painstakingly made all the original drawings in this work, and, finally, the J B Lippincott Company for their guidance and many suggestions in the production of this book.

A. F. DePALMA

Contents

1	ORIGIN AND COMPARATIVE ANATOMY OF THE PECTORAL LIMB	1
	Origin of Paired Appendages	1
	Evolution of the Pectoral Girdle	2
	Fishes	2
	Amphibia	3
	Reptiles	3
	Birds	3
	Mammals	5
	Evolution of the Upper Extremities	5
	Scapula	6
	Humerus	9
	Muscles	11
2	NORMAL ANATOMY AND FUNCTIONAL MECHANISM OF THE SHOULDER JOINT AND CONGENITAL ABNORMALITIES	15
	Shoulder Joint	16
	Scapulohumeral (Glenohumeral) Joint	17
	Acromion Process	18
	Coracoid Process	18
	Glenoid Cavity	18
	Humerus	18
	Clavicle	20
	Articular Capsule of Scapulohumeral Joint	20
	Ligaments of the Shoulder Joint	23
	Coracohumeral Ligament	23
	Musculotendinous Cuff	24
	Coraco-acromial Ligament	24
	Bursae Around the Shoulder Joint	25
	Subacromial Bursa and Subcoracoid Bursa	25
	Sternoclavicular Joint	27
	Acromioclavicular Joint	27
	Terminology of Movements at Shoulder Joint	28
	Anatomic Position of the Extremity	28
	Elevation	28
	Abduction	29
	Forward Flexion	29
	Pivotal Position	29
	Dorsal Flexion	30
	Adduction	30
	Scapulohumeral Rhythm	30
	Motion at the Shoulder Joint	30
	Glenohumeral Joint Motion	30
	Sternoclavicular Joint Motion	31
	Acromioclavicular Joint Motion	31
	Practical Significance of Above Observations	31

2	NORMAL ANATOMY AND FUNCTIONAL MECHANISM (<i>Continued</i>)	
	Mechanical Force Requirements for Motion at the Shoulder Joint	32
	Abductor and Flexors of the Humerus	34
	Depressors of the Humerus	35
	Scapular Rotators	36
	Practical Significance of Muscle Force Couple Principle	38
	Gliding Mechanism of Biceps Tendon	39
	Impairment of Muscle Force Couple by Isolated Nerve Paralysis and Other Lesions	40
	Deltoid Paralysis	40
	Rupture of the Supraspinatus Tendon	41
	Rupture of the Musculotendinous Cuff	41
	Paralysis of Serratus Anterior	41
	Paralysis of Trapezius	42
	Ossification of the Upper End of the Humerus	44
	Congenital Abnormalities	45
	Congenital High Scapula (Sprengel's Deformity)	45
	Cleidocranial Dysostosis	49
	Humerus Varus	50
	Other Congenital Deformities of the Glenohumeral Joint	50
	Coracoclavicular Joint	50
	Congenital Dislocation of the Shoulder	50
3	VARIATIONAL ANATOMY AND DEGENERATIVE LESIONS OF THE SHOULDER JOINT	52
	Introduction	52
	Glenoid Side of the Glenohumeral Joint	53
	Materials and Methods	53
	Observations Referable to the Glenoid Cavity	55
	Labrum Glenoidale and Biceps Tendon	62
	Synovial Membrane	69
	Glenohumeral Ligaments and Synovial Recesses	73
	Middle Glenohumeral Ligament	74
	Superior Glenohumeral Ligament	76
	Inferior Glenohumeral Ligament	77
	Musculotendinous Cuff	77
	Bursal Side of the Glenohumeral Joint	80
	Subacromial Bursa	80
	Floor of the Bursa and the Outer Surface of the Rotator Cuff	81
	Tuberosities and Bicipital Groove	90
	Summary	93
	Lesions of the Shoulder Compatible with Good Function	94
	Introduction	94
	Materials and Methods	95
	Observations Referable to the Articular Cartilage of the Head of the Humerus	97
	Sulcus	100
	Synovial Membrane and Musculotendinous Cuff	100

Biceps Tendon	107
Discussion	108
4 RUPTURES OF THE MUSCULOTENDINOUS CUFF	112
Introduction	112
Ruptures (Tears) of the Musculotendinous Cuff	112
Incomplete and Complete Tears	113
History of Injury	114
Mechanism Responsible for Primary Tears of the Cuff	115
Incomplete Tears in the Supraspinatus Region of the Cuff	116
Complete Tears in the Supraspinatus Region of the Cuff	116
Clinical Features	119
Treatment	127
Conservative Treatment of Incomplete Tears	127
Conservative Treatment of Complete Tears	128
Surgical Treatment	128
5 FROZEN SHOULDER AND BICIPITAL SYNDROME	138
Historical Survey	138
Frozen Shoulder	140
Introduction	140
Clinical Features	140
Etiology and Pathogenesis	143
Treatment	146
Early Stages of the Disease	146
Later Stage of the Disease	146
Operative Technic	147
Manipulation for Frozen Shoulders	147
Case Reports	147
Bicipital Tenosynovitis	150
Introduction	150
General Observations	150
Bicipital Tenosynovitis Associated with Trauma	150
Bicipital Tenosynovitis Without Trauma	161
Coraco-acromial Ligament	168
Coracohumeral Ligament	168
Rupture of the Biceps Tendon	169
Proximal Tendon Ruptures	169
Traumatic Dislocation of the Tendon of the Long Head of the Biceps Brachii Muscle	172
6 CALCAREOUS TENDINITIS OF THE MUSCULOTENDINOUS CUFF	177
Site and Sex	177
Age	177
Occupation	178
Trauma	178
Infection and Constitutional Diseases	179
Pathogenesis	179

6	CALCAREOUS TENDINITIS OF THE MUSCULOTENDINOUS CUFF (<i>Continued</i>)	180
	Pathologic Alterations in the Subacromial Bursa and Musculotendinous Cuff	180
	Clinical Features	181
	Subacute Syndrome	181
	Acute Syndrome	182
	Chronic Syndrome	183
	Radiographic Characteristics	183
	Treatment	184
	Acute Syndrome	184
	Surgical Removal of the Calcareous Deposit	185
	Aspiration and Needling	187
	After Treatment	187
	Irrigation	188
	Manipulation	188
	Roentgen Ray Therapy	189
	Subacute and Chronic Syndrome	189
	Other Therapeutic Measures	190
7	DISLOCATIONS OF THE SHOULDER JOINT	192
	Dislocations of the Sternoclavicular Joint	192
	Treatment	192
	Dislocations of Acromioclavicular Joint	195
	Subluxation of Acromioclavicular Joint	195
	Complete Dislocation of Acromioclavicular Joint	196
	Treatment of Recent Lesions of Acromioclavicular Joint	198
	Treatment of Old Acromioclavicular Dislocations	203
	Dislocations of the Scapulohumeral Joint	204
	Acute Dislocations	204
	Mechanism of Production of Dislocation	207
	Pathology of Acute Anterior Dislocation	209
	Other Complications Exclusive of Fracture	212
	Fractures of the Humerus	214
	Clinical Features of Acute Anterior Dislocation	214
	Complications of Dislocation of the Shoulder	223
	Open Reduction for Initial Dislocation	223
	Luxatio Erecta	224
	Acute Traumatic Posterior Dislocation	224
	Old Dislocations	226
	Pathology	227
	Course of Untreated Cases	227
	Choice of Treatment	227
	Closed Methods	228
	Open Methods	228
	Recurrent Dislocation of the Glenohumeral Joint	229
	Pathology	230
	Pathogenesis of Recurrent Dislocation of the Shoulder	236
	Treatment	236
	Posterior Recurrent Dislocation	245
	Pathology and Treatment	245

8	FRACTURES AND FRACTURE DISLOCATIONS OF THE UPPER END OF THE HUMERUS	248
	General Considerations	248
	Mechanism of Fractures of the Upper End of the Humerus	249
	Classification of Fractures of the Upper End of the Humerus	252
	Fractures Requiring No Reduction	252
	General Considerations	252
	Management of Impacted Fractures of the Upper End of the Humerus	254
	Depressed Fracture of the Greater Tuberosity	254
	Impacted Fractures of the Anatomic Head into Greater Tuberosity	255
	Fractures Requiring Reduction	256
	Fractures of Greater Tuberosity with Displacement	256
	Fracture of the Lesser Tuberosity with Retraction	259
	Fractures of the Humeral Neck	260
	Surgical Neck Fractures with Complete Displacement	260
	Fractures of the Anatomic Neck of the Humerus	263
	Separation of the Humeral Epiphysis	264
	Treatment	265
	Fracture Dislocations of the Humerus	268
	Fracture of the Greater Tuberosity	268
	Fractures of the Head and the Neck of the Humerus, with Dislocation	270
9	SHOULDER PAIN OF NEUROGENIC ORIGIN AND OBSTETRIC PARALYSIS	274
	Introduction	274
	Referred Pain	275
	Anatomic Considerations	280
	Cervical Segment of the Spinal Column	280
	Brachial Plexus	282
	Topographic and Variational Anatomy of the Supraclavicular Region	285
	Syringomyelia	288
	Hypertrophic Cervical Pachymeningitis	289
	Platybasia (Basilar Compression)	290
	Herpes Zoster	290
	Inflammatory Lesions	291
	Guillain Barre Syndrome	291
	Lesions Associated with Pathologic Disorders of the Vertebral Column	291
	Tuberculosis	292
	Rheumatoid Arthritis	292
	Hypertrophic Arthritis	292
	Primary and Metastatic Neoplasms	293
	Traumatic Lesions	293
	Protrusion of the Intervertebral Disk	295
	Lesions Situated Outside the Vertebral Column	298
	Cervical Rib Syndrome	298
	Scalenus Anticus Syndrome	301
	Subcoracoid Pectoralis Minor Syndrome and Costoclavicular Syndrome	305
	Treatment	306
	Tumors	306
	Inflammatory Lesions	307
	Serum Neuritis	307

9	SHOULDER PAIN OF NEUROGENIC ORIGIN AND OBSTETRIC PARALYSIS (<i>Continued</i>)	
	Traumatic Lesions	308
	Obstetric Paralysis	308
	Introduction	308
	Etiology and Pathogenesis	309
	Treatment	310
	Types of Obstetric Paralysis	311
	Operative Procedures for Obstetric Paralysis	312
	Postoperative Management	313
	Osteotomy of the Humerus and Muscle Release	313
10	BONE TUMORS OF THE SHOULDER JOINT	316
	General Considerations	316
	Introduction	316
	Classification of Bone Tumors	316
	Diagnosis of Bone Tumors	317
	History	317
	Age of Patient	317
	Pain	318
	Injury	318
	Loss of Function	318
	Past History	318
	Site of Tumor	319
	Objective Findings	319
	Pulsating Tumors	320
	Body Reaction	320
	Roentgenographic Studies	321
	Laboratory Examinations	322
	Histologic Examination	323
	Giant-Cell Tumor	323
	Introduction	323
	Age and Sex	324
	Site	324
	Etiology	324
	Macroscopic Pathology	325
	Microscopic Pathology	325
	Clinical Features	327
	Physical Findings	328
	Roentgenologic Features	328
	Diagnosis	330
	Treatment	332
	Prognosis	334
	Preoperative and Postoperative Radiation	334
	Epiphyseal Chondromatous Giant Cell Tumor (Codman) [Benign Chondro- blastoma of Bone (Jaffe)]	335
	Introduction	335
	Origin	335
	Clinical Features	335
	Roentgenographic Features	335

Microscopic Features	336
Diagnosis	336
Treatment	336
Prognosis	337
Solitary Bone Cyst	337
Introduction	337
Origin	337
Age, Incidence and Site	338
Pathology	338
Clinical Features	340
Roentgenologic Features	340
Diagnosis	341
Treatment	341
Sarcomas of the Bone	341
Incidence and Age	341
Site	341
Gradation of Degree of Malignancy	341
Osteogenic Sarcoma	342
Sclerosing Osteogenic Sarcoma	342
Telangiectatic Sarcoma of the Bone (Osteolytic Type of Osteogenic Sarcoma or Malignant Bone Aneurysm)	343
Medullary and Subperiosteal Osteogenic Sarcoma	344
Fibrosarcoma of Bone	344
Ewing's Sarcoma	346
Introduction	346
Incidence Age Sex and Site	346
Clinical Features	347
Pathology	348
Prognosis and Treatment	348
Osteoid Osteoma	349
Introduction	349
Age Sex and Incidence	349
Site	349
Clinical Features	350
Roentgenologic Features	350
Pathology	351
Treatment	351
Chondrosarcoma	351
Introduction	351
Primary Chondroblastic Sarcoma	351
Secondary Chondrosarcoma	354
Chondroma	356
Incidence Site Age and Sex	356
Clinical Features	356
Roentgenographic Features	356
Treatment	357
Osteochondroma	357
Introduction	357
Etiology	357

9	SHOULDER PAIN OF NEUROGENIC ORIGIN AND OBSTETRIC PARALYSIS (<i>Continued</i>)	
	Traumatic Lesions	308
	Obstetric Paralysis	308
	Introduction	308
	Etiology and Pathogenesis	309
	Treatment	310
	Types of Obstetric Paralysis	311
	Operative Procedures for Obstetric Paralysis	312
	Postoperative Management	313
	Osteotomy of the Humerus and Muscle Release	313
10	BONE TUMORS OF THE SHOULDER JOINT	316
	General Considerations	316
	Introduction	316
	Classification of Bone Tumors	316
	Diagnosis of Bone Tumors	317
	History	317
	Age of Patient	317
	Pain	318
	Injury	318
	Loss of Function	318
	Past History	318
	Site of Tumor	319
	Objective Findings	319
	Pulsating Tumors	320
	Body Reaction	320
	Roentgenographic Studies	321
	Laboratory Examinations	322
	Histologic Examination	323
	Giant-Cell Tumor	323
	Introduction	323
	Age and Sex	324
	Site	324
	Etiology	324
	Macroscopic Pathology	325
	Microscopic Pathology	325
	Clinical Features	327
	Physical Findings	328
	Roentgenologic Features	328
	Diagnosis	330
	Treatment	332
	Prognosis	334
	Preoperative and Postoperative Radiation	334
	Epiphyseal Chondromatous Giant Cell Tumor (Codman) [Benign Chondro- blastoma of Bone (Jaffe)]	335
	Introduction	335
	Origin	335
	Clinical Features	335
	Roentgenographic Features	335

Incisions to Expose the Scapula	392
Exposure of Vertebral Border of Scapula	392
Arthrodesis of the Scapulohumeral Joint	395
General Considerations	395
Arthrodesing Procedures	399
Position of Optimum Function	402
Muscle Transplantations	404
Paralysis of the Deltoid Muscle	404
Transplantation of Fascial Extension of the Trapezius Muscle (Mayer)	405
Operative Technic	405
Postoperative Management	406
Transplantation of Biceps and Triceps Muscles (Ober)	406
Operative Technic	407
Transplantation of the Origin of the Posterior Portion of the Deltoid	409
Operative Technic (Harmon)	409
Fascial Transplants In Paralytic Disorders About the Shoulder Girdle	409
Paralysis of the Spinal and the Elevator Muscles of the Scapula	410
Operative Technic (Dickson)	410
Postoperative Management	410
Paralysis of the Serratus Anterior Muscle	410
Operative Technic (Dickson)	410
Paralysis of the Rhomboidei and the Levator Scapulae Muscles with Good	
Power in the Serratus Anterior Muscle	411
Operative Technic (Dickson)	411
Amputations	412
General Considerations	412
Amputation at the Level of the Surgical Neck	412
Disarticulation of the Shoulder	414
Interscapulothoracic Amputation	416
Resections	417
Excision of the Acromion Process	417
Resection of the Scapula	419
Resection of the Outer End of the Clavicle	420
Resection of the Inner End of the Clavicle	420
Total Excision of Clavicle	420
Resection of the Upper End of the Humerus	421
Resection of the Humeral Head	422

10 BONE TUMORS OF THE SHOULDER JOINT (<i>Continued</i>)	
Osteochondroma (<i>Continued</i>)	
Site Incidence Age and Sex	358
Clinical Features	358
Roentgenographic Features	359
Treatment and Prognosis	359
Multiple Myeloma	360
Introduction	360
Incidence, Age and Sex	360
Clinical Features	360
Pathology	362
Roentgenographic Features	362
Prognosis and Treatment	362
Solitary Plasma-Cell Myeloma	362
Metastatic Carcinoma in Bones of the Shoulder Girdle	362
Introduction	362
Types of Metastatic Bone Lesions	363
Pathologic Fractures	365
Roentgenographic Features	365
Myositis Ossificans Circumscripta	365
Introduction	365
Age Sex and Site	366
Clinical Features	366
Roentgenographic Features	366
Treatment	366
11 SURGICAL APPROACHES AND PROCEDURES	372
General Considerations	372
Anatomic Considerations	373
Principal Gliding Mechanisms	373
Deltoid Muscle	374
Cephalic Vein	375
Pectoralis Major	375
Bony Landmarks	375
Subacromial Bursa	376
Musculotendinous Cuff	376
Nerves	376
Arteries and Veins	378
Position of Patient	380
Anesthesia	381
Skin Incision	381
Approaches to Shoulder Joint Region	382
Anterior Approaches	382
Superior Approaches	386
Posterior Approaches	388
Approach to the Sternoclavicular Joint	390
Approach to the Acromioclavicular Joint (Infraclavicular Route of Cubbins et al)	390
Incision to Expose the Clavicle	392

Origin and Comparative Anatomy of the Pectoral Limb

ORIGIN OF PAIRED APPENDAGES EVOLUTION OF THE PECTORAL GIRDLE

FISHES
AMPHIBIA
REPTILES
BIRDS
MAMMALS

EVOLUTION OF THE UPPER EXTREMITIES

SCAPULA
HUMERUS
MUSCLES

ORIGIN OF PAIRED APPENDAGES

The origin of paired appendages has been the source of considerable controversy among morphologists. The lateral fin theory has supplanted the gill-arch theory of Gegenbaur and is now accepted as the most plausible explanation of the beginning of these appendages.

According to the lateral fin theory, paired limbs are derived from longitudinal lateral folds of epidermis extending backward along the body from just behind the gills to the anus. By accentuation of the anterior and the posterior and suppression and reduction of the intermediate portions of the folds, the pectoral and the pelvic fins were formed (Fig. 1). Into these folds, muscle buds migrated from the ventral border of the adjoining myotomes, giving rise to radial muscles which motivated the fins and were the forerunners of the intrinsic muscles of the hand (Bunnell). The muscle buds disclosed a metameric arrangement and derived their nerve supply from ventral roots of the spinal nerves.

Peripheral nerve fibers in the base of the fin divide repeatedly, giving rise to a complex plexus. The number of myotomes which comprise the muscular apparatus of the fin

is disclosed by the number of spinal nerves which contribute to the plexus. In ontogeny, motor nerves always supply the muscles for which they were designed originally. Muscles exhibiting a nerve supply from more than one spinal nerve denote combining of muscular tissue of several segments. Next in the process of evolution of the appendages was the appearance of radials (cartilage rays) between the muscle buds; these provided more strength and support to the fins (Fig. 2).

Concentration and fusion of the proximal (basal) ends of the radials in the fin gave rise to the basilia (basal cartilages) which extended inward into the body wall to form the most primitive girdle (Fig. 3). In order to meet the requirements of a freely movable fin, an articulation appeared in the basal plates. Further evolution of the girdle includes fusion of the basilia of either side in the midline to form a ventral bar, also included is a dorsal extension of the arch above the level of the articulation to join the axial skeleton. Thus a complete girdle is formed around the body. The above steps in the ontogeny of the girdle have been noted in the *Selachia* (elasmobranchs) and also in *Chondrostei* and *Teleostei*.

Origin and Comparative Anatomy of the Pectoral Limb

ORIGIN OF PAIRED APPENDAGES

EVOLUTION OF THE PECTORAL GIRDLE

FISHES

AMPHIBIA

REPTILES

BIRDS

MAMMALS

EVOLUTION OF THE UPPER EXTREMITIES

SCAPULA

HUMERUS

MUSCLES

ORIGIN OF PAIRED APPENDAGES

The origin of paired appendages has been the source of considerable controversy among morphologists. The lateral fin theory has supplanted the gill arch theory of Gegenbaur and is now accepted as the most plausible explanation of the beginning of these appendages.

According to the lateral fin theory paired limbs are derived from longitudinal lateral folds of epidermis extending backward along the body from just behind the gills to the anus. By accentuation of the anterior and the posterior and suppression and reduction of the intermediate portions of the folds the pectoral and the pelvic fins were formed (Fig. 1). Into these folds muscle buds migrated from the ventral border of the adjoining myotomes giving rise to radial muscles which motivated the fins and were the forerunners of the intrinsic muscles of the hand (Bunnell). The muscle buds disclosed a metameric arrangement and derived their nerve supply from ventral roots of the spinal nerves.

Peripheral nerve fibers in the base of the fin divide repeatedly giving rise to a complex plexus. The number of myotomes which comprise the muscular apparatus of the fin

is disclosed by the number of spinal nerves which contribute to the plexus. In ontogeny motor nerves always supply the muscles for which they were designed originally. Muscles exhibiting a nerve supply from more than one spinal nerve denote combining of muscular tissue of several segments. Next in the process of evolution of the appendages was the appearance of radials (cartilage rays) between the muscle buds, these provided more strength and support to the fins (Fig. 2).

Concentration and fusion of the proximal (basal) ends of the radials in the fin gave rise to the basilla (basal cartilages) which extended inward into the body wall to form the most primitive girdle (Fig. 3). In order to meet the requirements of a freely movable fin an articulation appeared in the basal plates. Further evolution of the girdle includes fusion of the basilla of either side in the midline to form a ventral bar, also included is a dorsal extension of the arch above the level of the articulation to join the axial skeleton. Thus a complete girdle is formed around the body. The above steps in the ontogeny of the girdle have been noted in the *Selachia* (elasmobranchs) and also in *Chondrostei* and *Teleostei*.

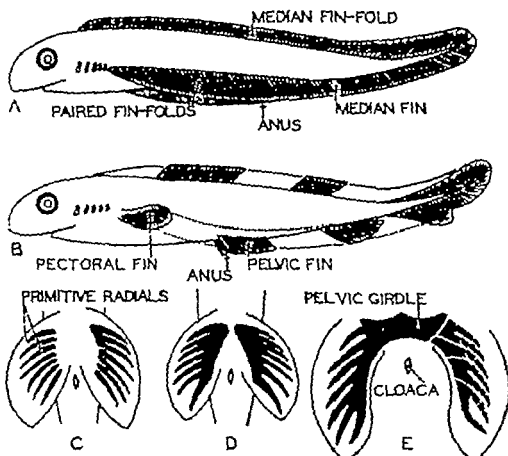


FIG. 1 Hypothetical evolution of paired fins and their skeletal supports. (A) Primitive stage characterized by continuous fin folds: the dorsal and ventral fins posterior to the anus are median and unpaired. (B) Elasmobranch stage: paired fin folds persist only in the region of the pectoral and pelvic fins; median fins have become discontinuous. (C-E) Hypothetical stages in the evolution of the skeleton of the pelvic fins of elasmobranch fishes. The right side of C and E represent a later stage in the phylogenesis than the left. E represents the differentiated skeletons of the girdle and the extremity (after Wiedersheim) (Neal and Rand, *Chordate Anatomy*, Philadelphia: Blakiston).

EVOLUTION OF THE PECTORAL GIRDLE

FIGURES

In its basic pattern the girdle is an inverted arch spanning the ventral surface of the body and extending dorsally on either side above the level of the articulation. Both the girdle and the limb are free. Each girdle comprises a ventral segment (coracoid) and a dorsal segment (scapula). These at the point of juncture form the glenoid fossa which articulates with the basal component of the skeleton of the limb. Further segmentation of the scapula gives rise to the suprascapula which may become attached

to the axial skeleton (as in *skates*). All the above elements have separate centers of chondrification (Fig. 4).

Further in the scale of evolution of the pectoral girdle is the appearance of a girdle of membranous bones derived from the skin. It encircles the head starting from behind the gills. The elements of either half of the girdle join and fuse in the midline on the ventral surface of the body through the medium of the interclavicle. Each half of this membranous circle consists of four membranous bones: (1) posttemporal which is jointed with the skull; (2) supraclathrum; (3) clathrum; and (4) clavicle.

The interclavicle which unites the girdle

ventrally is an unpaired bone. Both the basal girdle and membranous girdle eventually became attached to one another. Such is the basic plan of the pectoral girdle as noted in two genera (*Eusthenopteron* and *Sauripterus*) of the upper Devonian crossopterygians. These are considered the ancestors of the amphibia whose appendages possessed the pattern which made the evolution of the tetrapod limb possible (Fig 5)

AMPHIBIA

With the attainment of terrestrial habits most of the elements of the membranous girdle (posttemporal and supracleithrum) decreased in size and disappeared while the cartilaginous girdle began to assume a more significant role. The skull was freed of all attachment to the girdle. In urodeles all vestiges of the membranous girdle have disappeared.

In the amphibia the tripartite type of pectoral girdle made its first appearance, the coracoid represented by the ventral bar in the fishes became segmented into the anterior procoracoid and posterior coracoid while the clavicle came in relation to the procoracoid. No significant alterations occur in the suprascapula and the scapula. A noteworthy observation in the pectoral girdle of large amphibia (*Rhachitomi*) is the direction of the glenoid fossa. It faces laterally indicating that the humerus extended away from the trunk in the coronal plane horizontal to the ground. Its articular surface was screw shaped (Howell) indicative of clumsy arm movement.

REPTILES

Whereas in the amphibia the pectoral girdle is just behind the head in the reptile it has migrated a considerable distance from this position. Essentially, the girdle comprises a scapula, a procoracoid and a coracoid. In general the clavicle replaces the procoracoid as evidenced by the latter's reduction in size. However in some reptiles the clavicle is absent (*Crocodylia* and *Chamaeleo*). Some reptiles lost their limbs

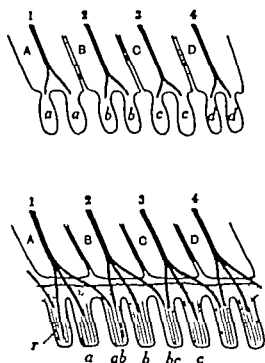


FIG 2 Formation of adult radial muscles from embryonic muscle buds and their motor nerve supply. Above, embryonic stage with a pair of buds to each segment; below, adult stage with radial muscles compounded of material from adjacent buds 1-4, four spinal nerves A-D, four myomeres a-d, muscle buds r, radial muscle (Goodrich E. S. *Studies on the Structure and Development of Vertebrates*, London: Macmillan, p. 134).

and the girdles are either greatly reduced or have disappeared (*Amphisbaenines*, *Ophidia*).

BIRDS

Elements of the girdle of the reptiles were modified in birds to permit flight. The clavicles exhibit a marked degree of development, their ventral ends fusing to form the wishbone (furcula). The scapula is small, curved and narrow, extending backward. The coracoid is large and strong, one end together with the scapula forming the glenoid fossa while the other unites with the sternum. The keeled sternum provides attachment for the strong pectoral muscles used in flight. In some cursorial birds the clavicles are greatly reduced (emu), while in others they are absent.

MAMMALS

In monotremes the lowest order of mammals, large coracoids are found between the sternum and the glenoid fossa. In all other mammals, however, the coracoid tends to become greatly reduced, forming an insignificant process on the scapula. The only other vestige of the bone is the coracoid ligament, extending from the coracoid process to the bone, in which may be found isolated masses of cartilage. It has a separate center of ossification. This arrangement frees the scapula from any bone attachment to the skeleton. In mammals without clavicles the scapula has no bony attachments whatsoever. It becomes the sole support for the limb and provides attachment for muscles necessary for a freely movable extremity. New functional demands on the girdle resulted in the development of a projection of bone on the dorsal surface of the scapula (spina scapulae) which extends downward and ends in the acromion.

Generally the clavicle articulates with the acromion and the sternum, its only connection to the coracoid process being by the coracoclavicular ligaments (conoid, trapezoid). In mammals which have acquired freedom of the forelimb to a marked degree such as insectivores, primates and some marsupials and rodents the clavicle is

usually well developed. In others, including ungulates, carnivores, cetaceans and some rodents, edentates and marsupials it is absent or rudimentary.

EVOLUTION OF THE UPPER EXTREMITIES

There has been considerable controversy as to the derivation of the cheiropterygium (tetrapod limb, also called the pentadactyl limb) from the ichthyopterygium (paired fins of fishes). It was recorded previously that in the evolution of the free paired appendages the proximal or basal ends of the radials (cartilage rays) fused to form basilia and later with the demand of greater movability of the fin a joint appeared between the radials and the basilia, several of which in turn articulated with the girdle. Such a scheme is discernible in the paired fins of the elasmobranchs, which possess three basilia (propterygium, mesopterygium and metapterygium) located between the girdle and the radials of the fin (Fig. 4).

In the pectoral girdles and the fins of the crossospts, *Eusthenopteron* and *Sauripterus* (fossils from upper Devonian) is found an arrangement of the skeletal elements generally accepted as a link between paired fins of fishes and tetrapod limb (Fig. 5). These two genera of crossopterygian fishes are considered close to the forms from

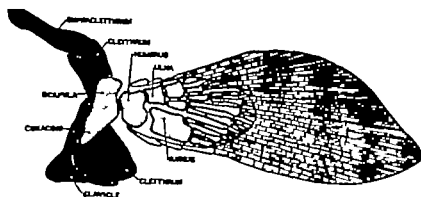


FIG. 5. Diagram of reconstructed pectoral girdle and fin of *Sauripterus* and upper Devonian crossopterygian fish. It exhibits a close similarity of relations of proximal elements of the extremity to those found in the pectoral extremity of tetrapods (redrawn from Brown) (Neal and Rand, *Chordate Anatomy*, Philadelphia, Blakiston).

MAMMALS

In nontrimes, the lowest order of mammals large coracoids are found between the sternum and the glenoid fossa. In all other mammals however the coracoid tends to become greatly reduced forming an insignificant process on the scapula. The only other vestige of the bone is the coracoid ligament extending from the coracoid process to the bone in which may be found isolated masses of cartilage. It has a separate center of ossification. This arrangement frees the scapula from any bone attachment to the skeleton. In mammals without clavicles the scapula has no bony attachments whatsoever. It becomes the sole support for the limb and provides attachment for muscles necessary for a freely movable extremity. New functional demands on the girdle resulted in the development of a projection of bone on the dorsal surface of the scapula (spina scapulae) which extends downward and ends in the acromion.

Generally the clavicle articulates with the acromion and the sternum its only connection to the coracoid process being by the coracoclavicular ligaments (conoid trapezoid). In mammals which have acquired freedom of the forelimb to a marked degree such as insectivores, primates and some marsupials and rodents the clavicle is

usually well developed. In others including ungulates, carnivores, cetaceans and some rodents, edentates and marsupials it is absent or rudimentary.

EVOLUTION OF THE UPPER EXTREMITIES

There has been considerable controversy as to the derivation of the cheiropterygium (tetrapod limb also called the pentadactyl limb) from the ichthyopterygium (paired fins of fishes). It was recorded previously that in the evolution of the free paired appendages the proximal or basal ends of the radials (cartilage rays) fused to form basilia and later with the demand of greater movability of the fin a joint appeared between the radials and the basilia several of which in turn articulated with the girdle. Such a scheme is discernible in the paired fins of the elasmobranchs which possess three basilia (propterygium, mesopterygium and metapterygium) located between the girdle and the radials of the fin (Fig. 4).

In the pectoral girdles and the fins of the crossospts *Eusthenopteron* and *Sauripterus* (fossils from upper Devonian) is found an arrangement of the skeletal elements, generally accepted as a link between paired fins of fishes and tetrapod limb (Fig. 5). These two genera of crossospterygian fishes are considered close to the forms from

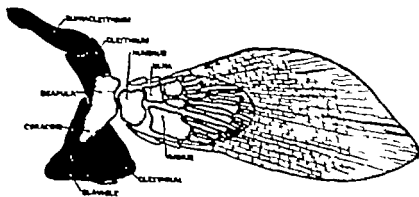


FIG. 5 Diagram of reconstructed pectoral girdle and fin of *Sauripterus* and upper Devonian crossospterygian fish. It exhibits a close similarity of relations of proximal elements of the extremity to those found in the pectoral extremity of tetrapods (redrawn from Brown) (Neal and Rand Chordate Anatomy Philadelphia Blakiston)

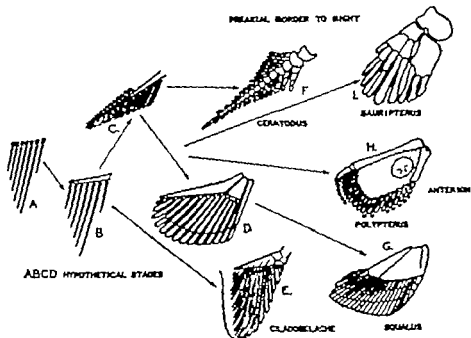


FIG 3 Diagrams illustrating hypothetical evolution of the extremities of diapnoan (I) ganoid (H) and elasmobranch (G) from a fin fold supported by a series of similar radial cartilages. By fusion of radial cartilages basilia (basal cartilages) are formed. Skeletal supports of the fins eventually differ in relation of the basal elements to the radialis (Redrawn from A. Brazier Howell) (Neal and Rand Chordate Anatomy Philadelphia, Blakiston)

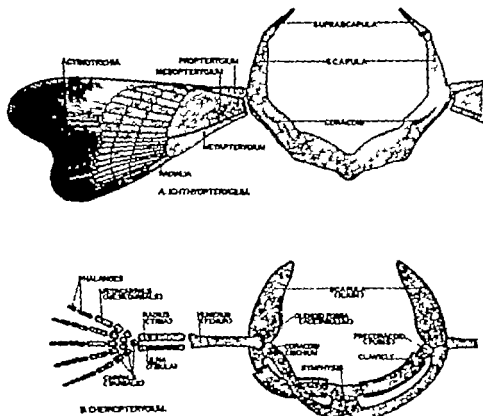


FIG 4 Diagrams illustrating scheme of pectoral appendages of lower and higher vertebrates (Bottom) Names of corresponding parts of pelvic appendages are shown in parentheses. (Neal and Rand Chordate Anatomy Philadelphia Blakiston)

shifting from a position directed laterally to one directed posteriorly and inferiorly. As a result of the change in posture, the coracoid's function decreased. Hence, a gradual reduction in its size is noted in this group. Up to this stage in evolution of the pectoral girdle no evidence of a spine on the dorsal surface of the scapula is found except in the Therapsida whose posture is not unlike that of the mammals.

Posture was responsible for the development of the scapular spine which is found in all mammals except the very primitive forms the Monotremata. With rearrangement of some and disappearance of other muscles the need of a procoracoid and coracoid no longer existed. Therefore the former element disappeared entirely, while the latter was reduced to the coracoid process. The shape of the scapula is dependent upon posture and the functional requirements of the muscles attached to it. It is

broad and massive in forms which need large powerful serratus anticus muscles to support heavy bodies in a quadruped position.

In mammals which have partially or completely freed the pectoral limbs, the shape of the scapula exhibits a trend toward the pattern found in man. These alterations are brought about by change in posture from the pronograde to the orthograde and highly specialized functional requirements of a prehensile limb. The most significant scapular change is in the relation of length to breadth of the bone. Pronograde forms disclose a long narrow scapula, while in the ascent toward man it becomes broader.

This morphologic change is most obvious in the primates. That portion of the scapula below the spine demonstrates the most pronounced alterations, those in the region above the spine being insignificant. Morphologic modifications in the scapula can

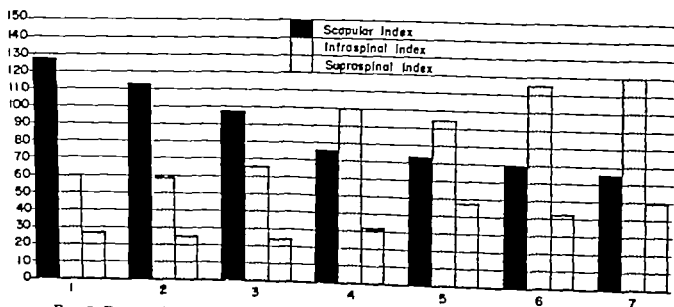
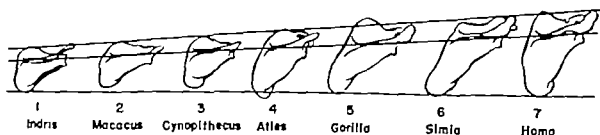


FIG 7 Progressive decrease in scapular index in successive stages from the pronograde to the orthograde (Redrawn from Inman Saunders and Abbott J Bone & Joint Surg 26 2)

which the amphibia evolved. The basic pattern of their pectoral limb comprised a proximal segment which was jointed to two middle segments which in turn articulated with several distal elements. The proximal element was destined to become the humerus, the middle elements the radius and the ulna, the distal elements the carpus and the digits.

The change from an aqueous to a terrestrial existence was accompanied by pronounced alteration in the skeletal elements of the pectoral fin which now must be used for support and locomotion. Therefore in the amphibia, the first animals to adopt terrestrial habits, the pentadactyl limb evolved from the paired fins. From the distal element arose the carpus, the meta carpus and the phalanges. The principal element in the radial side became the thumb and those on the ulnar side the other four digits. In all stages of evolution up to and including man the basic plan of the pentadactyl limb was maintained.

SCAPULA

During the evolution of the upper extremity, the scapula more than any other bone of the shoulder girdle reflects momentous alterations that have been brought about by increased functional demands of a prehensile limb. Changes in posture provided the stimulus which initiated the numerous morphologic changes. In the amphibia the scapula was high in the cervical region but was freed from the skull. Rhachitomous amphibians possessed massive scapulae with the glenoid cavity pointing laterally. The articulating surface was screw-shaped and the limbs were held in the coronal plane horizontal to the ground.

In the Reptilia the scapula with the entire girdle migrated a great distance from the skull in order to permit a more efficient mode of locomotion. The scapula was still broad and massive in the primitive forms. However later with increased efficiency in locomotion there was a trend toward reduction of this bone, the glenoid cavity

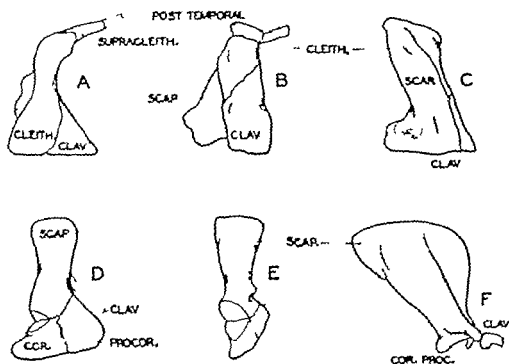


FIG. 6. Phylogenesis of the pectoral girdle. (A) *Sauripterus* (Devonian crossopterygian lung fish). (B) *Eogyrinus* (Carboniferous embolomereous amphibian). (C) *Eryops* (Permian rhachitomous amphibian). (D) *Moschops* (Permian dinoccephalian reptile). (E) *Cynognathus* (Triassic theriodont reptile). (F) *Macaca* (an Old World Recent monkey). (Howell *Speed in Animals*, University of Chicago Press p. 138.)

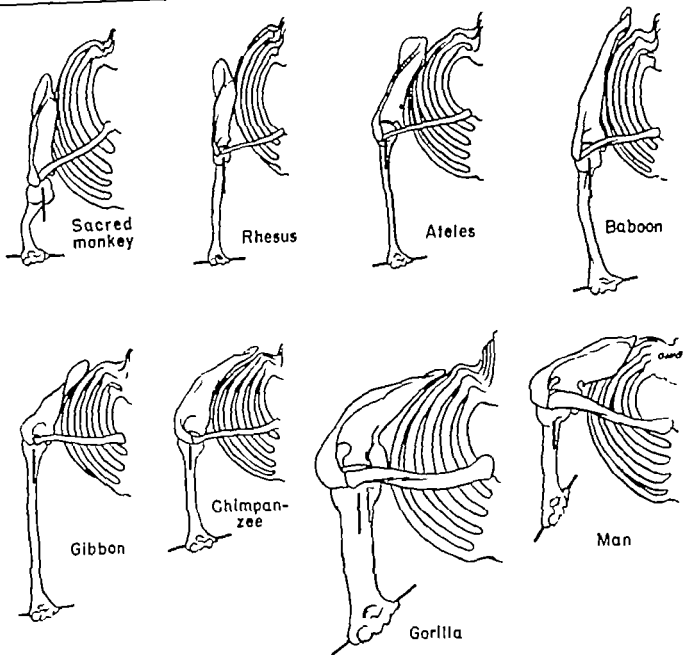


FIG 9 Changes in the thoracic cage the scapula and the humerus, in successive stages from the pronograde to the orthograde. The thoracic cage shows flattening in the antero-posterior plane and the scapula migrates to a dorsal position so that the glenoid cavity is directed laterally. The humerus shows a progressive increase in the torsion angle.

nence of the outer end of the spine the acromion process. Whereas in pronograde forms the acromion process is insignificant, in orthogrades it is a massive structure overlying the humeral head (Fig 8).

HUMERUS

During evolution of a prehensile extremity profound morphologic modifications occurred in the humerus. In rhachitomous amphibians the humerus was a massive bone flattened at either end the

distal end being larger than the proximal to provide attachment for large forearm muscles. In reptiles with free motion in the forelimb the upper extremity was brought beneath the body, and the humerus became less massive. Two nodules appeared at the proximal end which evolved into the tuberosities of the mammalian humerus. The anterior became the greater and the posterior the lesser tuberosity.

Generally speaking in mammals adapted for running (ungulates—horse) the articu-

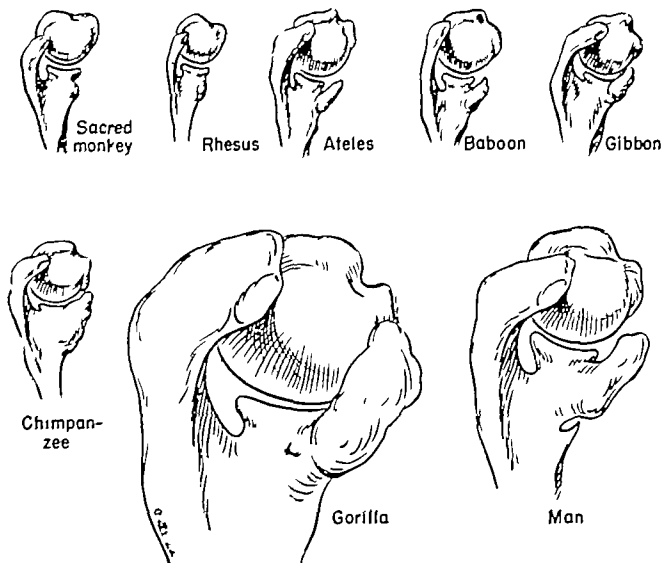


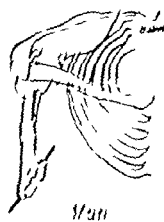
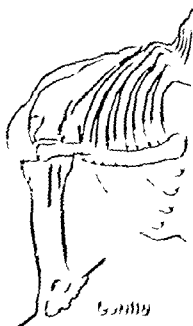
FIG 8 Gradual increase in spine of the scapula and the acromion process during development from the pronograde to the orthograde. This change reflects the increasing importance of the deltoid muscle. Also note the increase in size of the coracoid process the inequality of the two tuberosities of the head of the humerus and the inner displacement of the intertubercular sulcus in successive stages of development

be expressed by a scapular index, a ratio of the breadth (measured along the base of the spine) to the length (measured from the superior to the inferior angle) The scapular index is high in the pronograde in which the scapula is long, narrow and slender The index progressively decreases in the successive stages of development approaching man (orthograde)

This is the result of a gradual increase in the breadth of the scapula and elongation of the bone below the level of the spine giving rise to a progressive increase in the

"infraspinous index (Fig 7) Inman Saunders and Abbott, in their comprehensive study of the function of the shoulder joint observed that lengthening of the scapula below the spine changed the relation of the axillary border of the scapula to the glenoid fossa, thereby altering the angle of pull of the muscles attached to this region a feature of great significance in the mechanism of the shoulder

In the primates as one approaches man, the increasing importance of the role of the deltoid muscle is reflected in the promi



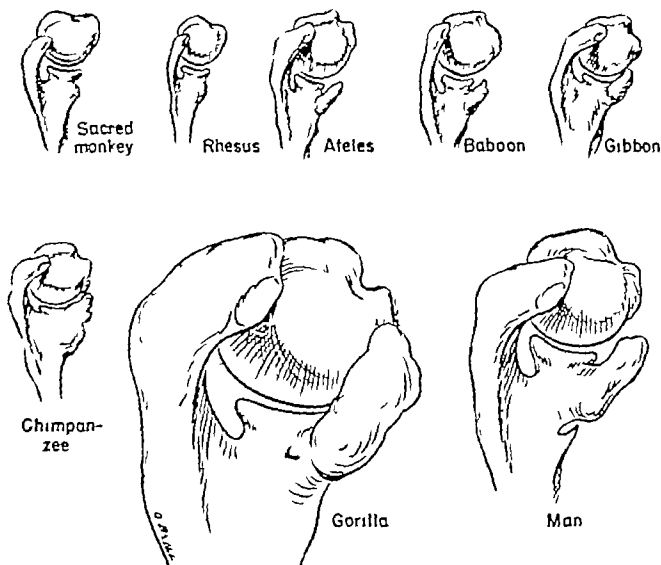


FIG. 8 Gradual increase in spine of the scapula and the acromion process during development from the pronograde to the orthograde. This change reflects the increasing importance of the deltoid muscle. Also note the increase in size of the coracoid process the inequality of the two tuberosities of the head of the humerus and the inner displacement of the intertubercular sulcus in successive stages of development.

be expressed by a "scapular index" a ratio of the breadth (measured along the base of the spine) to the length (measured from the superior to the inferior angle). The scapular index is high in the pronograde in which the scapula is long narrow and slender. The index progressively decreases in the successive stages of development approaching man (orthograde).

This is the result of a gradual increase in the breadth of the scapula and elongation of the bone below the level of the spine giving rise to a progressive increase in the

"infraspinous index" (Fig. 7). Inman Saunders and Abbott in their comprehensive study of the function of the shoulder joint observed that lengthening of the scapula below the spine changed the relation of the axillary border of the scapula to the glenoid fossa thereby altering the angle of pull of the muscles attached to this region a feature of great significance in the mechanism of the shoulder.

In the primates as one approaches man, the increasing importance of the role of the deltoid muscle is reflected in the promi-

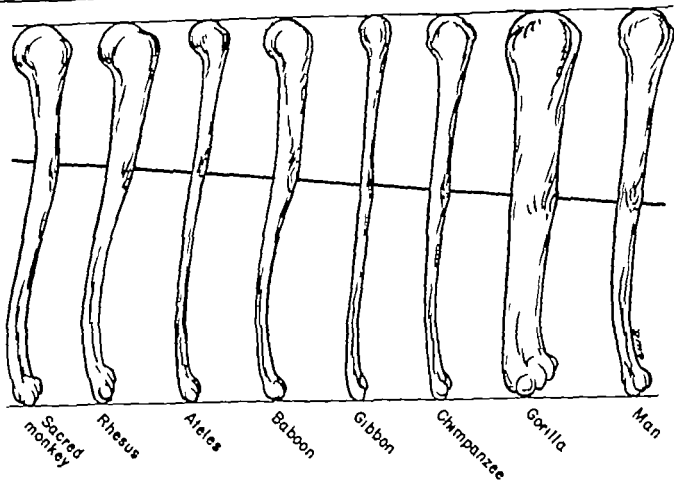


FIG 11 Deltoid insertion migrates progressively to a lower level on the shaft of the humerus, indicating the significant role played by the deltoid in higher primates.

to a more distal position. This feature together with increase in size of the acromion, greatly increases the leverage of the deltoid muscle (Fig 11).

Other significant morphologic alterations were recession of the lesser tuberosity and medial displacement of the bicipital groove. Pronograde forms disclose the biceps tendon passing over the center of the head of the humerus and entering the groove in the same plane. In this position it acts as a strong elevator of the arm. Both tuberosities in these forms are approximately the same size.

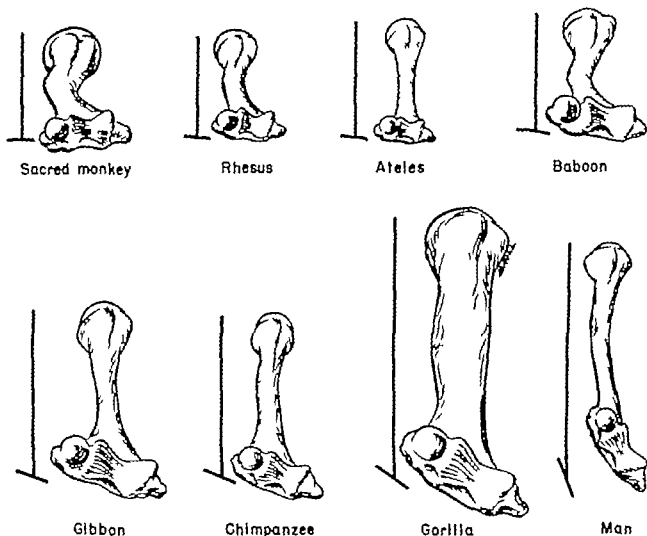
A different relationship is found in orthogrades. In these forms the bicipital groove has been rotated medially by torsion of the humerus so that a line passing through the center of the head of the humerus in man makes an angle of 30° with one passing through the plane of the groove (Inman, Saunders and Abbott). Marked reduction in

the size of the lesser tuberosity is a characteristic feature in the higher primates.

From the above observations it is obvious that the biceps tendon (long head) functions at a greater mechanical disadvantage, further increased by using the arm in a position of internal rotation. In this position the biceps tendon plays over the medial wall of the groove and the lesser tuberosity now really functions as a trochlea.

MUSCLES

Changes in posture and functional requirements of a prehensile extremity were responsible for alterations in the topography and the morphology of muscles about the shoulder. Such changes were primarily responsible for the skeletal modifications previously indicated. The extent of the change in any individual muscle becomes apparent when its relative mass is compared with the total mass of the group in



PROGRESSIVE INCREASE IN TORSION IN SHAFT OF HUMERUS

FIG 10 Progressive increase in torsion of the humerus resulting in inward rotation of the bicipital groove. The articular surfaces at either end of the humerus rotate in opposite directions.

lar surface of both ends of the humerus function in the same plane (sagittal plane) a line passing through the long axis of the head of the humerus is directed forward and one through the distal articular surface transversely. Meeting of these two axes describes a torsion angle of 90° . In primates as the orthograde form is approached, the torsion angle increases. Man discloses some variation in the torsion angle. Australians exhibit an angle of 134° and the French and the Swiss 164° (Martin 1928).

Several factors are responsible for the changing relationship of the articular surfaces of the humerus. Development of the

orthograde forms was accompanied by an teroposterior flattening of the thoracic cage and dorsal displacement of the scapula. The glenoid fossa is now directed laterally (Fig 9). Prehensile requirements however demand that the extremity as a whole function anterior to the body and that the elbow be maintained in the parasagittal plane. To meet these specifications the humeral shaft twists inwardly while the articular surfaces at either end rotate in the opposite directions (Fig 10). The dominant role acquired by the deltoid in the higher primates is demonstrated further by the progressive shift of the deltoid insertion on the humerus

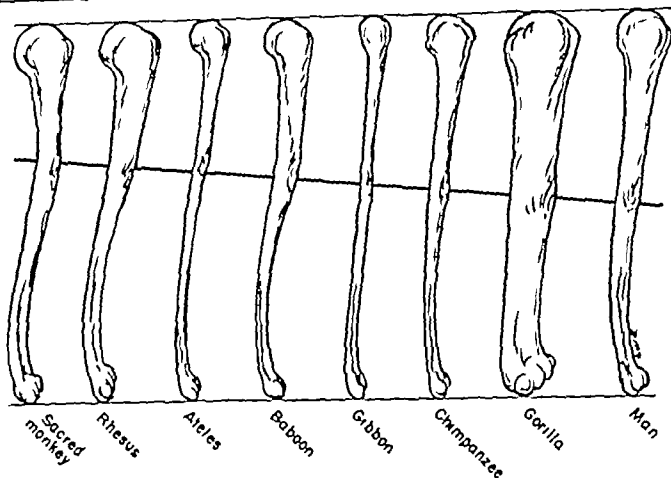


FIG 11 Deltoid insertion migrates progressively to a lower level on the shaft of the humerus indicating the significant role played by the deltoid in higher primates

to a more distal position. This feature together with increase in size of the acromion greatly increases the leverage of the deltoid muscle (Fig 11).

Other significant morphologic alterations were recession of the lesser tuberosity and medial displacement of the bicipital groove. Pronograde forms disclose the biceps tendon passing over the center of the head of the humerus and entering the groove in the same plane. In this position it acts as a strong elevator of the arm. Both tuberosities in these forms are approximately the same size.

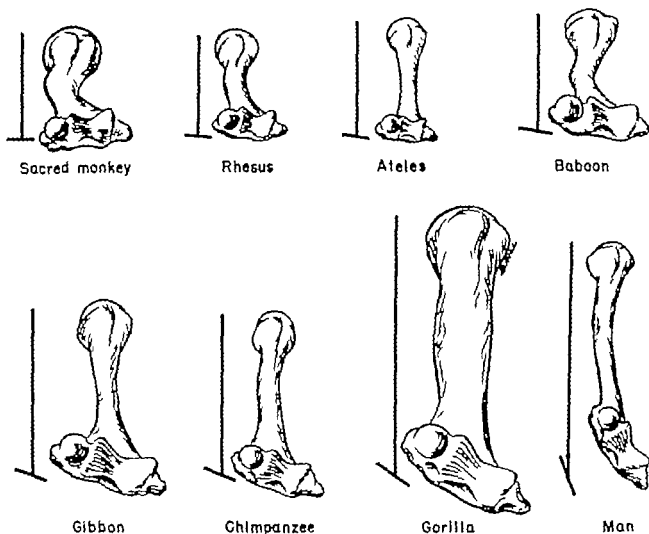
A different relationship is found in orthogrades. In these forms the bicipital groove has been rotated medially by torsion of the humerus so that a line passing through the center of the head of the humerus in man makes an angle of 30° with one passing through the plane of the groove (Inman, Saunders and Abbott). Marked reduction in

the size of the lesser tuberosity is a characteristic feature in the higher primates.

From the above observations it is obvious that the biceps tendon (long head) functions at a greater mechanical disadvantage further increased by using the arm in a position of internal rotation. In this position the biceps tendon plays over the medial wall of the groove and the lesser tuberosity now really functions as a trochlea.

MUSCLES

Changes in posture and functional requirements of a prehensile extremity were responsible for alterations in the topography and the morphology of muscles about the shoulder. Such changes were primarily responsible for the skeletal modifications previously indicated. The extent of the change in any individual muscle becomes apparent when its relative mass is compared with the total mass of the group in



PROGRESSIVE INCREASE IN TORSION IN SHAFT OF HUMERUS

FIG 10 Progressive increase in torsion of the humerus resulting in inward rotation of the bicipital groove. The articular surfaces at either end of the humerus rotate in opposite directions.

lar surface of both ends of the humerus function in the same plane (sagittal plane) a line passing through the long axis of the head of the humerus is directed forward and one through the distal articular surface transversely. Meeting of these two axes describes a torsion angle of 90° . In primates as the orthograde form is approached the torsion angle increases. Man discloses some variation in the torsion angle. Australians exhibit an angle of 134° and the French and the Swiss 164° (Martin 1928).

Several factors are responsible for the changing relationship of the articular surfaces of the humerus. Development of the

orthograde forms was accompanied by an teroposterior flattening of the thoracic cage and dorsal displacement of the scapula. The glenoid fossa is now directed laterally (Fig 9). Prehensile requirements however, demand that the extremity as a whole function anterior to the body and that the elbow be maintained in the parasagittal plane. To meet these specifications, the humeral shaft twists inwardly while the articular surfaces at either end rotate in the opposite directions (Fig 10). The dominant role acquired by the deltoid in the higher primates is demonstrated further by the progressive shift of the deltoid insertion on the humerus

which it belongs. Following the scheme of Inman, Saunders and Abbott, the muscles which partake in shoulder mechanism can be categorized into three topographic units (1) scapulohumeral group (2) axiohumeral group and (3) axioscapular group

The study made on the functional mechanism of the shoulder by the aforementioned workers is so complete, comprehensive and logical that one is forced to draw heavily from this source of information when discussing this topic. Many of their observations are noted in the subsequent section.

THE SCAPULOHUMERAL GROUP These concern the scapula to the humerus and consist of the supraspinatus, infraspinatus, teres minor, subscapularis and deltoid muscles. Concurrently with acquisition of a free limb the relative deltoid mass increases while that of the supraspinatus decreases. Forty-one per cent of the total mass of this unit in man is made up by the deltoid muscle.

Comparative anatomy further discloses that the teres minor muscle is wanting in early mammals and that it evolved from the deltoid to form a separate muscle passing from the inferior angle of the scapula to the humerus. With elongation of the infraspinatus portion of the scapula the relative mass of this muscle progressively increased until in man it makes up 5 per cent of the total mass. Although it is a morphologic component of the deltoid, the teres minor, because of topographic changes, plays an entirely different role in the mechanism of the shoulder than that of the deltoid.

The subscapularis muscle is little affected by morphologic alterations from the primitive to the higher primates. It makes up 20 per cent of the mass of the scapulohumeral group. The only significant alteration is an increase in number of fasciculi of origin. This is the result of elongation of the scapula. This same skeletal change brought about an increase in the area of attachment of the infraspinatus which con-

stitutes approximately 16 per cent of the total mass.

According to Inman, Saunders and Abbott, the last three muscles (subscapularis, teres minor and infraspinatus), by reason of alterations in the morphology and the topography of the group and the elongation of the scapula, function as a unit. They are both rotators and depressors of the head of the humerus.

The axioscapular group chiefly concerned with the mechanism of the shoulder, comprises (1) serratus anterior, (2) rhomboids, (3) levator scapulae and (4) trapezius muscles. The first three muscles of this unit originated from one complex of muscle fibers arising from the ribs (first eight or ten) and their homologues (transverse processes of the cervical vertebrae) in the cervical region and inserting into the vertebral border of the scapula. In primitive forms the dominant function of this group was to control the movements of the vertebral border of the scapula.

In general, those fibers concerned with dorsal motion of the scapula became the rhomboid muscles; those with ventral motion the serratus muscle; and those with cranial displacement of the scapula the levator scapulae. Function and posture were responsible for evolution of the individual muscle as they exist in the higher primates. The serratus anterior formed the basal unit for all three muscles. Concentration of the proximal and distal fibers and progressive reduction of the intermediate fibers gave origin to two distinct muscles, the levator scapulae and the serratus anterior.

Further morphologic alterations in the serratus anterior comprise grouping of its proximal and distal fibers, progressive reduction in size of its intermediate fibers, and insertion of the dominant upper and lower portions of the muscle into the superomedial and inferior angles of the scapula.

The trapezius, like the sternocleidomastoid muscle, evolved from a muscle sheet passing from the last gill arch to the membranous girdle. In terrestrial forms it at-

tained a position from the occipital region to the trunk in tetrapods it arises from the occiput, the middorsum of neck and thorax, and inserts into the spine of the scapula the acromion and the scapula. Little change has occurred in the trapezius in the evolution of the primates. There has been, however, some concentration of its proximal and distal muscle components and reduction in mass and efficiency of its middle components.

The axiohumeral group is made up of the pectoralis major, the pectoralis minor and the latissimus dorsi muscles and extends from the trunk to the humerus. The pectoral group evolved from a primitive muscle sheet which connected the coracoid with the humerus. Change in posture and increased functional demands made on the limb were responsible in the later reptilian and early mammalian forms for displacement of part of this muscle sheet dorsally to gain attachment to the scapula which later gave rise to the suprapinatus, the infrapinatus and the anterior part of the subcapularis. All other components of the muscle migrated from the procoracoid to the sternum and gave rise to the pectoralis major.

Further morphologic modification in the pectoralis major resulted in a division of this mass into a superficial and a deep layer. Part of the sternal attachment of the superficial fibers shifted forward and gained attachment to the clavicle (clavicular head of the pectoralis major). From the deep layer evolved the pectoralis minor muscle which in higher primates discloses its humeral attachment in primitive forms to have migrated to the coracoid process.

The latissimus dorsi and teres major muscles originate from a single basic muscle sheet extending from the trunk caudal to the scapula to the humerus. They demonstrate in the higher primates no significant

morphologic or topographic alterations except that they are unusually well developed in forms specializing in climbing.

Biceps Brachii and Triceps Muscles. Both these muscles evolved from ventral and dorsal brachial muscle elements which were concerned primarily with motion in the more distal joints, the elbow and the wrist. From the ventral brachial elements arose the biceps muscle by proximal migration along a fascial plane of brachial components to reach the scapula (Howell). In mammals other than primate it is a single muscle. Cursorial forms (horse) disclose powerful biceps which together with the suprapinatus act as a single functional unit to elevate the foreleg.

Primates exhibit two heads of origin, one from the supraglenoid tubercle and the other from the coracoid process. Medial displacement of the bicipital groove resulting from torsion of the humeral shaft places the long head at a mechanical disadvantage thereby losing its efficiency as an elevator of the arm which it possesses in other forms. However, the biceps can be made to function as an abductor of the extremity if the arm is rotated externally, hence restoring the tendon to the top and the center of the humeral head. This maneuver is not infrequently utilized by individuals with paralyzed abductors of the arm.

The triceps originated from a dorsal brachial muscle element. Like the biceps its three heads migrated proximally. The scapular or long head gained attachment on the infraglenoid tubercle, the medial head to the upper and posteromedial surface of the humerus and the lateral head to the upper posterolateral surface. No significant morphologic or topographic alterations have occurred in this muscle. It functions as a powerful extensor (dorsal flexor) of the arm.

which it belongs. Following the scheme of Inman, Saunders and Abbott, the muscles which partake in shoulder mechanism can be categorized into three topographic units: (1) scapulohumeral group, (2) axiohumeral group and (3) axioscapular group.

The study made on the functional mechanism of the shoulder by the aforementioned workers is so complete, comprehensive and logical that one is forced to draw heavily from this source of information when discussing this topic. Many of their observations are noted in the subsequent section.

THE SCAPULOHUMERAL GROUP These connect the scapula to the humerus and consist of the supraspinatus, infraspinatus, teres minor, subscapularis and deltoid muscles. Concurrently with acquisition of a free limb the relative deltoid mass increases, while that of the supraspinatus decreases. Forty-one per cent of the total mass of this unit in man is made up by the deltoid muscle.

Comparative anatomy further discloses that the teres minor muscle is wanting in early mammals and that it evolved from the deltoid to form a separate muscle passing from the inferior angle of the scapula to the humerus. With elongation of the infraspinatus portion of the scapula the relative mass of this muscle progressively increased until in man it makes up 5 per cent of the total mass. Although it is a morphologic component of the deltoid the teres minor, because of topographic changes, plays an entirely different role in the mechanism of the shoulder than that of the deltoid.

The subscapularis muscle is little affected by morphologic alterations from the primitive to the higher primates. It makes up 20 per cent of the mass of the scapulohumeral group. The only significant alteration is an increase in number of fasciculi of origin. This is the result of elongation of the scapula. This same skeletal change brought about an increase in the area of attachment of the infraspinatus which con-

stitutes approximately 16 per cent of the total mass.

According to Inman, Saunders and Abbott, the last three muscles (subscapularis, teres minor and infraspinatus), by reason of alterations in the morphology and the topography of the group and the elongation of the scapula function as a unit. They are both rotators and depressors of the head of the humerus.

The axioscapular group chiefly concerned with the mechanism of the shoulder, comprises (1) serratus anterior (2) rhomboids, (3) levator scapulae and (4) trapezius muscles. The first three muscles of this unit originated from one complex of muscle fibers arising from the ribs (first eight or ten) and their homologues (transverse processes of the cervical vertebrae) in the cervical region and inserting into the vertebral border of the scapula. In primitive forms the dominant function of this group was to control the movements of the vertebral border of the scapula.

In general, those fibers concerned with dorsal motion of the scapula became the rhomboid muscles; those with ventral motion the serratus muscle; and those with cranial displacement of the scapula, the levator scapulae. Function and posture were responsible for evolution of the individual muscle as they exist in the higher primates. The serratus anterior formed the basal unit for all three muscles. Concentration of the proximal and distal fibers and progressive reduction of the intermediate fibers gave origin to two distinct muscles: the levator scapulae and the serratus anterior.

Further morphologic alterations in the serratus anterior comprise grouping of its proximal and distal fibers, progressive reduction in size of its intermediate fibers, and insertion of the dominant upper and lower portions of the muscle into the superomedial and inferior angles of the scapula.

The trapezius like the sternocleidomastoid muscle evolved from a muscle sheet passing from the last gill arch to the membranous girdle. In terrestrial forms it at-

tained a position from the occipital region to the trunk, in tetrapods it arises from the occiput, the middorsum of neck and thorax, and inserts into the spine of the scapula the acromion and the scapula. Little change has occurred in the trapezius in the evolution of the primates. There has been, however, some concentration of its proximal and distal muscle components and reduction in mass and efficiency of its middle components.

The axiohumeral group is made up of the pectoralis major, the pectoralis minor and the latissimus dorsi muscles and extends from the trunk to the humerus. The pectoral group evolved from a primitive muscle sheet which connected the coracoid with the humerus. Change in posture and increased functional demands made on the limb were responsible in the later reptilian and early mammalian forms for displacement of part of this muscle sheet dorsally to gain attachment to the scapula which later gave rise to the supraspinatus, the infraspinatus and the anterior part of the sub-capularis. All other components of the muscle migrated from the procoracoid to the sternum and gave rise to the pectoralis major.

Further morphologic modification in the pectoralis major resulted in a division of this mass into a superficial and a deep layer. Part of the sternal attachment of the superficial fibers shifted forward and gained attachment to the clavicle (clavicular head of the pectoralis major). From the deep layer evolved the pectoralis minor muscle which in higher primates discloses its humeral attachment in primitive forms to have migrated to the coracoid process.

The latissimus dorsi and teres major muscles originate from a single basic muscle sheet extending from the trunk caudal to the scapula to the humerus. They demonstrate in the higher primates no significant

morphologic or topographic alterations except that they are unusually well developed in forms specializing in climbing.

Biceps Brachii and Triceps Muscles. Both these muscles evolved from ventral and dorsal brachial muscle elements which were concerned primarily with motion in the more distal joints, the elbow and the wrist. From the ventral brachial elements arose the biceps muscle by proximal migration along a fascial plane of brachial components to reach the scapula (Howell). In mammals other than primates it is a single muscle. Cursorial forms (horse) disclose powerful biceps which together with the suprapinatus act as a single functional unit to elevate the foreleg.

Primates exhibit two heads of origin, one from the supraglenoid tubercle and the other from the coracoid process. Medial displacement of the bicipital groove resulting from torsion of the humeral shaft places the long head at a mechanical disadvantage thereby losing its efficiency as an elevator of the arm which it possesses in other forms. However, the biceps can be made to function as an abductor of the extremity if the arm is rotated externally, hence restoring the tendon to the top and the center of the humeral head. This maneuver is not infrequently utilized by individuals with paralyzed abductors of the arm.

The triceps originated from a dorsal brachial muscle element. Like the biceps its three heads migrated proximally. The scapular or long head gained attachment on the infraglenoid tubercle, the medial head to the upper and posteromedial surface of the humerus and the lateral head to the upper posterolateral surface. No significant morphologic or topographic alterations have occurred in this muscle. It functions as a powerful extensor (dorsal flexor) of the arm.

BIBLIOGRAPHY

- Ashley Montagu, F. M. *An Introduction to Physical Anthropology* Springfield Ill. Thomas 1947
- Bardeen C. R. and Lewis W. H. Development of limbs, body wall and back *Am J Anat.* 113:6 1901
- Bunnell S. *Surgery of the Hand*, ed. 2 Philadelphia, Lippincott 1948
- Goodrich, E. S. *Studies on the Structure and Development of Vertebrates* London Macmillan, 1930
- Gregory W. K. Present status of the problem of the origin of the tetrapod with special reference to the skull and paired limbs, *Ann New York Acad. Science* 26:317-383 1915
- Gregory W. K. Further observations on the pectoral girdle and fin of *Sauripterus Taylori* Hall, a crossopterygian fish from the upper Devonian of Pennsylvania with special reference to the origin of the pentadactylate extremities of tetrapods *Proc. Am. Philosoph. Soc.* 75:673 1935
- Howell A. B. *Speed in Animals* Chicago Univ Chicago Press, 1944
- Huxley T. H. *Anatomy of the Vertebrates* New York D Appleton & Co 1881
- Inman V. T., Saunders, J. B. deC. M., and Abbott L. C. Observations on the function of the shoulder joint, *J Bone & Joint Surg* 26:130 1944
- Jones F. W. Attainment of upright position of man, London, *Nature* 146:26-27 1940
- Kingsley J. S. *Comparative Anatomy of the Vertebrates*, Philadelphia, Blakiston, 1917
- Lewis W. H. The development of the arm in man, *Am J Anat.* 145:184 1902
- Minor R. W. The pectoral limb of Eryop and other primitive tetrapods *Bull. Am Museum Nat. Hist.* 51:145 1924-1925
- Neal, H. V. and Rang H. W. *Comparative Anatomy* Philadelphia, Blakiston, 1936

2

Normal Anatomy and Functional Mechanism of the Shoulder Joint and Congenital Abnormalities

SHOULDER JOINT

SCAPULOHUMERAL (GLENOHUMERAL) JOINT

LIGAMENTS OF THE SHOULDER JOINT

BURSAE AROUND THE SHOULDER JOINT

STERNOCLAVICULAR JOINT

ACROMIOCLAVICULAR JOINT

TERMINOLOGY OF MOVEMENTS AT SHOULDER JOINT

ANATOMIC POSITION OF THE EXTREMITY

SCAPULOHUMERAL RHYTHM

MOION AT THE SHOULDER JOINT

MECHANICAL FORCE REQUIREMENTS FOR MOTION AT THE SHOULDER JOINT

PRACTICAL SIGNIFICANCE OF MUSCLE FORCE COUPLE PRINCIPLE

GLIDING MECHANISM OF BICEPS TENDON

IMPAIRMENT OF MUSCLE FORCE COUPLE BY ISOLATED NERVE PARALYSIS AND OTHER LESIONS

CENTERS OF OSSIFICATION OF THE HEAD OF THE HUMERUS

CONGENITAL ANOMALIES

The accumulation of knowledge referable to the basic principles which govern the intricate mechanism of the shoulder makes it evident that the term "shoulder joint" needs clarification. Hitherto the term has been used loosely in many textbooks to designate the scapulohumeral articulation. Comprehensive analysis of the functional mechanism of the shoulder discloses, however, that the scapulohumeral articulation is only one component of a complex system which functions synchronously to produce precise, coordinated movements.

From a functional viewpoint, the shoulder joint comprises four distinct articulations: the scapulohumeral, the sternoclavicular, the acromioclavicular, and the scapulothoracic. Although the last structure is not a true joint anatomically, it must be considered as such functionally (Fig. 12).

Discussion of the anatomy of the shoulder joint should include more than a de-

scription of the topography of the numerous components. Any such discussion should embody the modern concepts of the functional mechanism of the shoulder, an understanding essential to the orthopedic surgeon in designing operative procedures to restore optimum function and power in a shoulder joint whose function has been severely impaired. Moreover, knowledge of the origin and the morphologic and topographic modifications that have occurred in the evolution of man's prehensile extremity facilitates comprehension of the basic principles involved in the above mechanism of function.

A discussion of this region would be incomplete without recording the numerous variations of normal anatomy of the inside of the scapulohumeral joint, recognition of which is imperative for the correct evaluation and therapy of pathologic processes commonly encountered in this region. These variations are of sufficient importance to

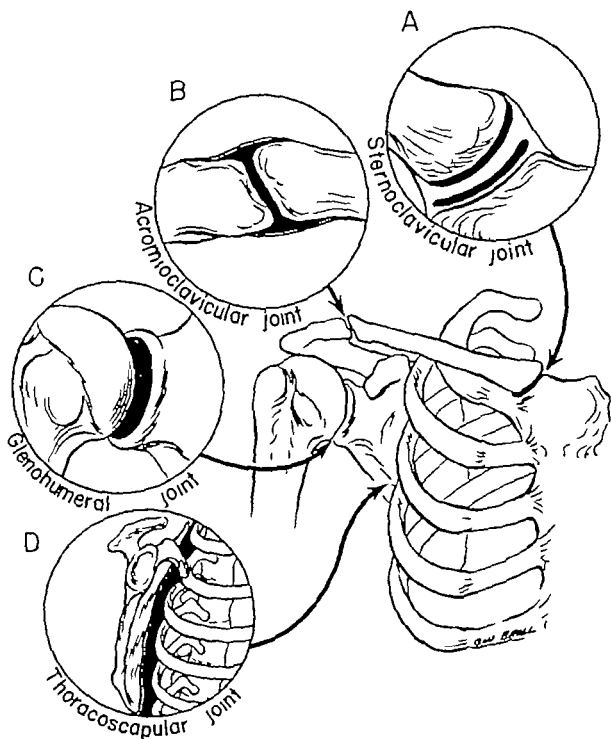


FIG. 12 Functional components of the shoulder joint. (A) Sternoclavicular joint. (B) Acromioclavicular joint. (C) Glenohumeral joint. (D) Thoracoscapular joint.

merit discussion in a separate chapter entitled *Variational Anatomy and Degenerative Lesions of the Shoulder Joint*.

SHOULDER JOINT

As previously noted the shoulder joint

from a functional viewpoint comprises four articulations (1) scapulohumeral (2) sternoclavicular (3) acromioclavicular and (4) scapulothoracic joints. All four perform simultaneously as a single unit, yet are capable of independent motion (Fig. 12).

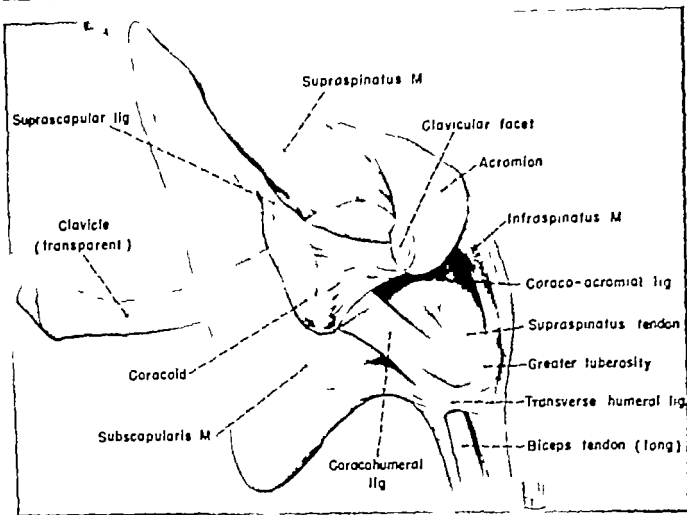


FIG. 13 Coraco-acromial arch overhanging the head of the humerus. Observe that the acromion is broad flat and massive; the oval clavicular facet faces obliquely upward and the triangular coraco-acromial ligament extends from the horizontal portion of the coracoid process to the tip of the acromion lateral to the clavicular facet.

SCAPULOHUMERAL (GLENOHUMERAL) JOINT

SCAPULA

The scapula has come to assume a most pertinent role in the functional mechanics of the shoulder joint. Topographic and morphologic alterations in its musculature are reflected in its size, configuration and position. It lies on the posterior surface of the thorax, suspended from the cervical and upper thoracic vertebrae by strong axio-scapular muscles (levator scapulae, rhomboids and upper digitations of both the trapezius and the serratus anterior muscles).

At rest with the arm at the side its superior angle is opposite the superior border

of the second rib, while its inferior angle is opposite the seventh intercostal space. It is almost completely enveloped in muscle masses. The ventral surface is slightly concave, conforming to the convex surface of the upper and posterior region of the thorax. Normally the scapula has a wide excursion over the posterior chest wall. It is free of any bony attachments to the axial skeleton except through the clavicle which serves as a strut between the scapula and the sternum. In man elongation or extension of the infraspinatus portion of the scapula has resulted in a decreased scapular index compared with that of other primates. On the other hand the infraspinatus index has increased (Fig. 7).

A triangular projection of bone, the spine

of the scapula, lies on the dorsal surface of the scapula. It is directed outward and slightly upward and terminates in the acromion process. The blade of the scapula is thin and delicate, while its bony projections (acromion, coracoid, spine head and neck) are strong.

ACROMION PROCESS

The spine of the scapula terminates in the acromion process, a prominent massive, flat bony projection which overhangs the humeral head from behind. Its superior flat surface slants outward backward and downward. The acromion articulates with the clavicle by means of an oval elongated articular facet facing obliquely upward. Anterior to the articular facet, the acromion receives the outer end of the coraco-acromial ligament (Fig. 13).

The acromion forms the outer bony component of the coraco-acromial arch. It is important to visualize the topographic anatomy of this bony process. As will be shown later, it plays a major role in the mechanism of shoulder movements, traumatic and degenerative lesions of the head of the humerus and the musculotendinous cuff and lesions of the subacromial bursa. Moreover, it protects the humeral head from impacts from behind and above.

In elevation of the arm the humeral head and tuberosities pass beneath the acromion. It is impossible to traumatize the humeral head directly unless a blow is directed to the front and the top of the shoulder while the arm is at the side and the elbow pulled backward (position of dorsal flexion). According to Grant, 21 per cent of adult scapulae exhibit failure of fusion of the acromial epiphysis with the acromion.

CORACOID PROCESS

The coracoid process is crooked, curved, stubby and bony and arises from the neck of the scapula just behind and to the inner side of the glenoid fossa. It is directed forward, outward and downward. Running transversely just above the coracoid is the

outer third of the clavicle, to which it is connected by the strong coracoclavicular ligaments (trapezoid and conoid ligaments). The coracoid process overhangs the humeral head anteriorly, as the acromion does posteriorly. The inner end of the coraco-acromial ligament is attached to the outer side of the upper aspect of the coracoid.

GLENOID CAVITY

This shallow fossa is shaped like an inverted comma. Its superior portion, which is designated the tail of the comma, is narrow, the inferior (head of the comma) is broad. It faces forward and upward and is covered by hyaline cartilage, which is thinner in the center of the head of the comma than at the outer margins of the articular cavity.

A fibrocartilaginous structure, the labrum glenoidale, triangular in cross section, surrounds the periphery of the glenoid cavity. In infant shoulders this structure is closely attached by its base to the brim of the glenoid cavity, its glenoid edge blending with the fibrils of the hyaline cartilage, while its capsular border is continuous with the fibrous capsule. In older decades the labrum in the region of the tail of the comma rests on the edge of the glenoid fossa like a meniscus, its glenoid border lying free (Fig. 14).

These anatomic features and their significance are discussed in detail in the following chapter. The long head of the biceps tendon inserts into the supraglenoid tubercle and is continuous with the posterior and anterior fibers of the labrum in the superior region of the tail of the comma (Fig. 14).

HUMERUS

The spherical head of the humerus rests on the shaft at an angle. Its articular surface is directed backward, medially and upward. Only a small portion of the head is in contact with the glenoid fossa at any one time during motion or at rest. Inward torsion of the shaft of the humerus has displaced the tuberosities and the bicipital groove medially.

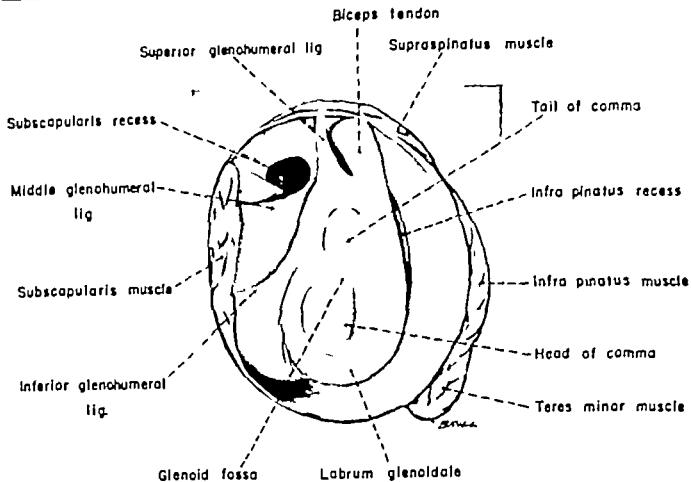


FIG. 14 The glenoid cavity is shaped like an inverted comma. Observe the superior, the middle and the inferior glenohumeral ligaments on the superior and the anterior aspects of the joint converging toward the biceps tendon. Also note the superior subscapularis recess (subscapularis bursa) situated immediately above the middle glenohumeral ligament. The biceps tendon blends with the fibers of the labrum glenoidale. A small recess in the posterior aspect of the joint forms the infraspinatus recess. It does not exist in all joints.

ally. The humeral torsion angle varies from 134° to 160° (Figs 9 and 10). The bicipital groove is displaced medially 30° from a line passing through the center of the head of the humerus. The greater tuberosity is directed externally forming the outer wall of the bicipital groove; the lesser tuberosity is directed forward and forms the inner wall of the groove. Between the tuberosities and the edge of the articular surface of the anatomic head is a broad sulcus called the anatomic neck of the humerus.

In living subjects the sulcus is obliterated by the musculotendinous cuff which completely fills this groove. It is very much in evidence in humeri stripped of all soft tissue structures. There is a gradual recession of this cuff from its line of insertion in

shoulders of individuals past middle life. It is not uncommon to see in these shoulders (when viewed from the inside of the joint) bare portions of the anatomic neck between the edge of the articular cartilage of the humeral head and the receding cuff. Such lesions were designated "rim rents" by Codman.

During elevation of the arm the greater tuberosity glides under the acromion or coraco-acromial arch with ease. However in lesions which produce hypertrophy of this bony process the tuberosity impinges on the acromion or the coraco-acromial arch thereby restricting the range of motion or causing a painful jog as it passes beneath the overlying structures. As will be shown in the discussion on motion at the shoulder

complete elevation can be achieved only in the frontal plane with the arm in internal rotation and in the coronal plane with the arm in external rotation

CLAVICLE

The clavicle is interposed between the acromion process and the sternum. It ex

maintaining the scapula in its normal dorsal position without the anterior bony strut. Through the joints at either end, the clavicle is capable of a wide range of motion which is essential to achieve complete elevation of the arm. The clavicle is bound to the coracoid process by the coracoclavicular ligaments, whose fibers permit considerable

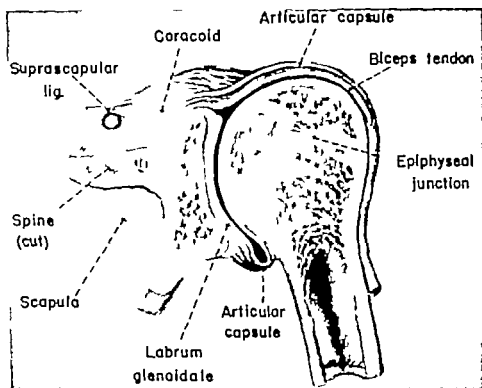


FIG. 15 Frontal section of the right shoulder joint, posterior view. Observe the redundancy of the capsule on the inferior aspect of the neck of the humerus. The fibrous capsule is lined with synovial membrane which is prolonged around the tendon of the long head of the biceps muscle as far as the surgical neck of the humerus providing a synovial cover for the osseofibrous bicipital tunnel. It then ends as a blind sac and is reflected upon the tendon. (Redrawn from S. W. Spalteholz, *Hand Atlas of Human Anatomy*, Philadelphia: Lippincott.)

hibits a double curve, being thick and cylindrical at the inner end and broad and flat at the outer end. It is generally believed to form a strut between the scapula and the axial skeleton, holding the upper extremity away from the body so that it can perform in the parasagittal plane. Nevertheless, it has been demonstrated clinically that resection of part or all of the clavicle does not allow a forward droop of the scapula.

One is forced to conclude that the axio-scapular group of muscles are capable of

rotation in its long axis. It provides attachment to the upper digitations of the trapezius, which elevates and supports the shoulder, and to the deltoid and the pectoralis major (clavicular head), which participates in elevation of the arm.

ARTICULAR CAPSULE OF SCAPULOHUMERAL JOINT

The fibrous capsule is a loose, redundant structure, twice the surface area of the humeral head (Fig. 15). Posteriorly and in

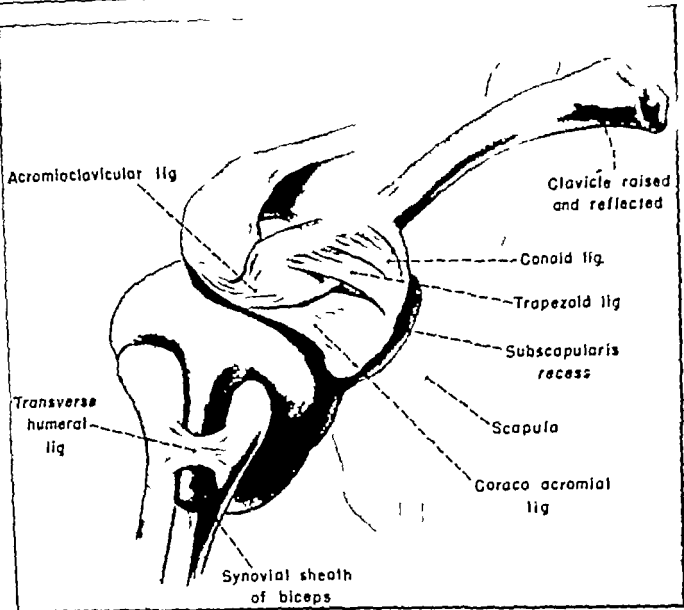


FIG 16 Extensions of the synovial capsule of the scapulohumeral joint and the coracoclavicular ligaments. Note the prolongation of the synovialis anteriorly under the coracoid process, forming the subscapularis recess and the second prolongation lining the bicipital groove it ends as a blind pouch and then is reflected over the biceps tendon.

teriorly it arises from the capsular border of the labrum glenoidale and the bone immediately adjacent to it. Anteriorly, its mode of origin depends upon the presence or absence and the size of the synovial recesses so commonly encountered in this region. When synovial recesses are present the capsule arises from the anterior neck of the scapula at varying distances from its articular surface. It becomes obvious, therefore that the presence and the size of the synovial recesses are determined by the distance from the articular surface of the scapula that the fibrous capsule arises from

the anterior neck of the scapula (Detailed anatomy of this region is discussed in Chapter 3.) In shoulder joints without synovial recesses the fibrous capsule arises from the entire circumference of the capsular border of the labrum glenoidale the rim of the glenoid cavity and the bone surrounding it.

Distally the fibrous capsule inserts superiorly into the upper portion of the anatomic neck and inferiorly into the periosteum of the humeral shaft at a considerable distance from the margin of the articular cartilage of the humeral head. The fibrous capsule is lined throughout by syno-

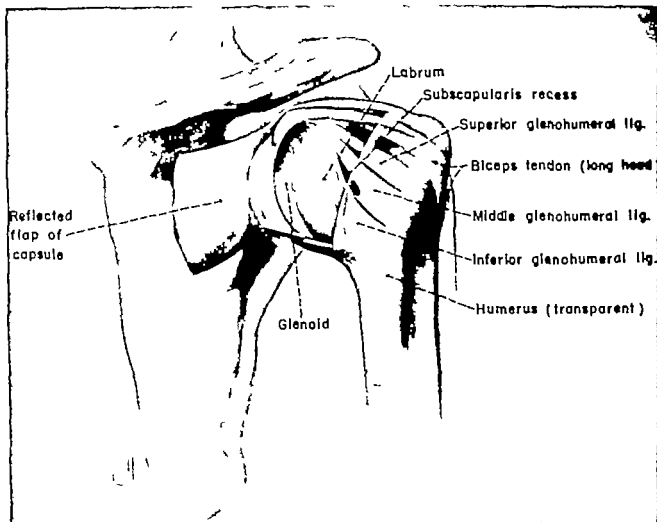


FIG 17 Interior of right shoulder joint, posterior view the posterior portion of the capsule has been reflected medially. Observe the arrangement of the three glenohumeral ligaments reinforcing the anterior aspect of the fibrous capsule. They are all directed toward the superior aspect of the glenoid fossa in this instance they all blend with the labrum glenoidale (not the case in all shoulders). Note the direct communication of the subscapularis recess with the inside of the joint cavity. In this joint the subscapularis recess communicates with the joint cavity both above and below the middle glenohumeral ligament.

vial membrane that is reflected inferiorly along the anatomic neck of the humerus toward the periphery of the articular cartilage of the humeral head blending with the hyaline cartilage of the head. Proximally it extends for varying distances over the labrum glenoidale and blends with its superficial fibers but fails to reach the articular cartilage of the glenoid cavity.

In the anterior region of the joint the synovial membrane is loose and redundant. It is prolonged (in 81.8 per cent of the shoulders) along the anterior surface of the

neck of the scapula toward the root of the coracoid process for varying distances to form the previously mentioned synovial recesses. It is also prolonged distally to line the bicipital groove and then reflected over the biceps tendon. The articular capsule is loose and redundant in the inferior region of the humeral neck. With the arm at the side the capsule forms a large nictitating fold in this region. As the arm is abducted this fold becomes smaller and disappears when full abduction is attained (Fig 16).

On all sides except the inferior portion,

the fibrous capsule is strengthened by the broad flat tendons of the rotator muscles (supraspinatus, infraspinatus, teres minor and sub-capularis muscles). These tendons are approximately 2.5 centimeters long

humeral head—the coracohumeral ligament and the musculotendinous cuff. The latter is not a true ligament but functionally it must be considered as such. Although the coracoclavicular ligaments are not related to a

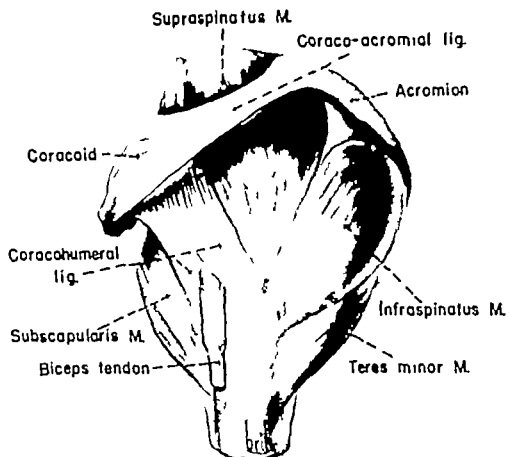


FIG. 18. Subdeltoid region of the left shoulder. Observe the coracohumeral ligament extending from the outer border of the horizontal limb of the coracoid process to both tuberosities bridging the intertubercular sulcus; also the triangular coraco-acromial ligament and the compactness of the rotator muscles which fuse with the fibrous capsule forming the musculotendinous cuff before they insert into the superior portion of the anatomic neck of the humerus. Note that the lower fibers of the subscapularis muscle anteriorly and the infraspinatus muscle and the teres minor posteriorly insert directly into the shaft of the humerus.

They blend with the fibrous capsule to form the musculotendinous cuff also referred to as the fibrotendinous cuff or the capsulotendinous cuff.

LIGAMENTS OF THE SHOULDER JOINT

About the shoulder are demonstrable ligamentous structures which are designed for different functions. Notably are those that provide a suspensory apparatus for the

joint; their integrity ensures stability of the shoulder girdle. The coraco-acromial ligament is concerned primarily with motion at the glenohumeral joint.

CORACOHUMERAL LIGAMENT

This ligament originates in the outer border of the horizontal limb of the coracoid process, passes forward and downward in the interval between the supraspinatus and the subscapularis muscles. Here its

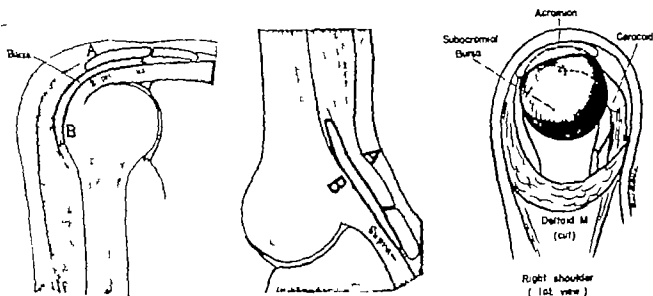


FIG 19 (*Left and Center*) Coronal sections through the right shoulder. Observe that the roof of the bursa comprises the acromion process and the subdeltoid fascia and its base overlies the tuberosity and the supraspinatus tendon (*Center*) The position of the bursa is depicted with the arm elevated, note that the greater tuberosity has passed beneath the acromion and point B has passed to point A (Codman *The Shoulder*, p 25) (*Right*) Superolateral view of the right shoulder with the deltoid removed note the spherical shape of the bursa and its extension under the acromion and inward toward the coracoid process. Occasionally it extends beneath the coracoid process.

fibers interlace with those of the fibrous capsule and insert with the capsule into both tuberosities bridging the bicipital groove. In this position it acts as a suspensory ligament of the humeral head. Its most proximal fibers are so arranged that they unwind and elongate upon external rotation of the shaft of the humerus functioning as a checkrein to external rotation. The ligament shortens upon internal rotation (Fig 18). This fiber arrangement is significant in the frozen shoulder. In this lesion as the result of a diffuse inflammatory process the fibers become fixed in the shortened position and help freeze the head in a position of internal rotation.

Occasionally, the ligament is continuous with the tendon of the pectoralis minor running between the two limbs of the coraco-acromial to reach the fibrous capsule of the glenohumeral joint.

MUSCULOTENDINOUS CUFF

All four short rotator muscles comprise this structure (supraspinatus, infraspinatus,

teres minor and subscapularis muscles). The muscles end in broad flat tendons whose fibers fuse with those of the fibrous capsule. So complete is the interlacing of tendon and capsular fibers that it is impossible to separate the two structures by sharp dissection. Microscopic examination of the distal one-half to three-quarters inches of the musculotendinous cuff fails to distinguish between the tendinous and capsular components of the cuff which inserts into the upper one-half of the anatomic neck of the humerus completely filling the sulcus. In this position it is apparent that this structure functions as a suspensory ligament for the humeral head (Fig 18).

CORACO-ACROMIAL LIGAMENT

This triangular structure forms a strong arch across the coracoid and the acromion. It arises with a wide base from the outer edge of the coracoid and tapers to a narrow band to insert into the inner border of the acromion just in front of the acromioclavicular joint. Its central fibers are very thin

and often wanting, forming essentially two strong limbs, an anterior and a posterior limb which join at their point of insertion into the acromion (Fig 18)

Considerable significance is attached to the coraco-acromial structure. It separates the subacromial bursa from the acromioclavicular joint, and its under surface forms the roof of the posterior part of the above bursa. In elevating the limb, the tuberosities pass beneath the arch. Codman believed that in elevating the arm this structure guided the head of the humerus and prevented it from gaining a fulcrum on the acromioclavicular joint. On the contrary, however it has been demonstrated that the integrity of this structure may be sacrificed with impunity without noticeable effect on the functional mechanism of the joint provided that the muscular apparatus is intact. The ligament or segments of it are routinely severed or removed without unfavorable sequelae.

The coraco-acromial arch with the underlying subacromial bursa and loose areolar tissue provides a gliding mechanism between the deep and the superficial muscle strata. Thus it becomes clear why lesions disturbing this mechanism or impingement of the tuberosities or a hypertrophied subacromial bursa against the coraco-acromial arch interfere with the smooth rhythmic elevation of the arm. Such lesions are discussed in their appropriate chapters.

All other ligaments about the shoulder have been described in the discussion pertaining to their respective joints. The glenohumeral ligaments and labrum glenoidale are taken up in Chapter 3. Variational Anatomy and Degenerative Lesions of the Shoulder Joint.

BURSAE AROUND THE SHOULDER JOINT

SUBACROMIAL BURSA AND SUBCORACOID BURSA (Fig 19)

The complex arrangement of the muscles which overlay the scapulohumeral joint comprise two muscular sleeves: an inner

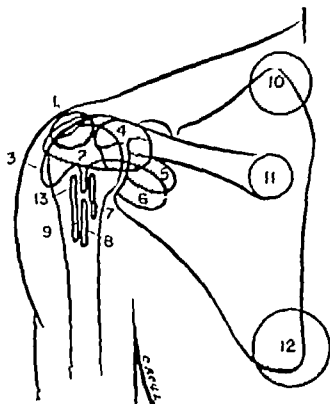


FIG 20 Normal bursae of the shoulder joint (1) subcutaneous acromialis (2) subacromial bursa (3) subdeltoid bursa (4) subcoracoid bursa (5) bursa of infraspinatus muscle (6) subscapular bursa, (7) bursa of latissimus dorsi, (8) bursa of teres major. Note 2, 3 and 4 are essentially one bursa; occasionally they may be separated by thin movable film of tissue. 5, 6 and 13 are prolongations of the synovial capsule (13 lines the bicipital groove). 9, 10, 11 and 12 are inconstant and have no official anatomic names. (Redrawn from Codman *The Shoulder*, p. 29)

sleeve made up of the short rotator muscles and an outer sleeve of the deltoid and the teres major muscles. These muscular sleeves operate one within the other—a performance made possible by an efficient gliding mechanism between the two strata. Fine filmy, areolar tissue and the subacromial bursa constitute the gliding mechanism. So efficiently does this arrangement function that often it is referred to as a secondary scapulohumeral joint.

The subacromial bursa adheres firmly by its base to the upper and outer portions of the greater tuberosity and to the outer surface of the musculotendinous cuff. Its roof is adherent to the under surface of the

acromion and to the under surface of the coraco-acromial ligament. Its lateral walls are prolonged loosely downward under the deltoid muscle, backward and outward under the acromion and mesially under the coracoid process.

Therefore as Codman pointed out it is

1 Bursae beneath tendons near their insertion into the humerus on either side of the bicipital groove as seen under the pectoralis major, the latissimus dorsi and the teres major muscles.

2 Supracoracoid bursa—most likely to be found when there is an anomalous inser-

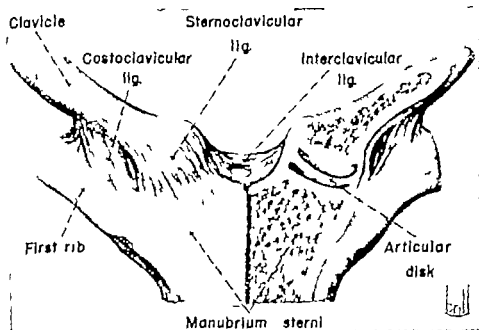


FIG. 21 Coronal section of the left sternoclavicular joint. Observe that the joint is divided into two compartments by the fibrocartilaginous disk. The articular end of the clavicle and the sternum are covered by fibrocartilage instead of hyaline cartilage. The interclavicular and the costoclavicular ligaments add to the stability of the joint. (Redrawn from S. W. Spalteholz, *Hand Atlas of Human Anatomy*, Philadelphia, Lippincott.)

apparent that the bursa in the subcoracoid area is part of the subacromial bursa. The subacromial bursa communicates with the joint cavity only if a tear involving the complete thickness of the musculotendinous cuff opens into the floor of the bursa.

The other important bursal structures—the subscapularis bursae (recesses) and the infraspinatus bursae (which really are extensions or diverticulae of the glenohumeral joint cavity beneath the respective tendons)—are discussed in detail in Chapter 3.

Numerous other bursae of lesser significance have been described about the shoulder region and may be listed as follows (Fig. 20):

tion of the pectoralis minor into the coracoid process.

3 *Infraserratus bursa*—located between the inferior angle of the scapula and the thoracic wall (Terillon).

4 *Subscapular bursa*—situated between the upper anterior region of the scapula and the upper three ribs (Goldthwait).

The last two bursae have been credited with being responsible for painful crepitation in the posterior region of the shoulder when the scapula is in motion, particularly during such elevation of this bone as shrugging the shoulder.

5 *Supra-acromial bursa* (subcutanea acromialis) situated between the skin and the

dorsum of the acromion, with its base fixed to the acromion

6 Bursa between insertion of the trapezius and the dorsum of the base of the spine

STERNOCLAVICULAR JOINT

This joint is formed by the mesial end of the clavicle and the facet on the posterolateral surface of the manubrium sterni. It is the only articulation between the trunk and the upper extremity. The configuration of the articulating surfaces of the clavicle and the sternum provides no stability to the joint; its integrity depends solely on the surrounding capsule, ligaments and articular disk.

The joint cavity is divided into two separate compartments by a fibrocartilaginous disk whose periphery is thicker than its center. Directed upward and outward the articular disk anchors the clavicle to the first rib by strong fibers which connect its upper border to the clavicle and its lower border to the first costal cartilage. This articular disk plays a very significant role in the mechanism of the shoulder. It acts as a shock absorber, protecting the sternum from the full impact of forces directed toward the trunk through the upper extremity.

The articular surfaces of the joint are surrounded by an articular capsule which is attached also to the edges of the interarticular fibrocartilage. Both anterior and posterior surfaces of the fibrous capsule are reinforced by strong fibrous bands running upward and outward constituting the anterior and posterior sternoclavicular ligaments.

Further stability to the joint is provided by the interclavicular and the costoclavicular or rhomboid ligaments. Fibers of the interclavicular ligament run from the top of one clavicle to the other and fuse with the capsular fibers of the joint. It is strong yet allows considerable range of motion of the clavicles. The strong spiral fibers of the costoclavicular ligament which unwind when the shoulder is brought upward and

backward run upward and outward between the first costal cartilage and the proximal end of the clavicle (outside of the sternoclavicular joint).

Modern concepts of mechanism of shoulder movement emphasize the important role contributed by the sternoclavicular joint. Also as will be demonstrated the integrity of the articulation must be maintained to attain complete shoulder movement. Its obliteration results in definite impairment of shoulder function.

ACROMIOCLAVICULAR JOINT

The lateral end of the clavicle demonstrates a shallow surface directed downward and outward, superimposed on the flattened elongated facet of the acromion to form the acromioclavicular articulation. While occasionally in a sagittal plane the joint is usually in an oblique plane. This arrangement permits upward displacement of the clavicle end when the stabilizing ligaments of the joint are disrupted. It is obvious that the articular surfaces favor instability rather than stability of the articulation, whose integrity is maintained entirely by the weak capsular ligaments and the strong coracoclavicular ligaments (trapezoid and conoid ligaments) (Fig. 16).

Occasionally a rudimentary interarticular wedge-shaped disk is present with its base attached to the superior portion of the articular fibrous capsule. In most instances it divides only the upper half of the joint cavity; rarely does it extend the entire width. Integrity of the joint is provided chiefly by the coracoclavicular ligaments which arise from the coracoid process, are continuous posteriorly and diverge anteriorly to form the outer trapezoid and inner conoid ligaments. They are directed upward and backward to insert into the clavicle.

Although the above structures firmly anchor the clavicle to the coracoid process, their fibers are so arranged that a relative lengthening of the ligaments occurs when the clavicle is rotated on its long axis. This

anatomic arrangement makes possible 20° of the total range of upper extremity abduction at the acromioclavicular joint.

TERMINOLOGY OF MOVEMENTS AT SHOULDER JOINT

It is necessary to adopt a group of terms which will best express the numerous movements of the extremity made possible by

joint because it occupies a unique position in relation to the planes of the body. Also all of its movements are summations of movements occurring simultaneously in all its component joints. Figure 22 depicts the anatomic planes through or in relation to which the upper extremity moves at the shoulder joint. It must be understood that in all movements of the extremity all

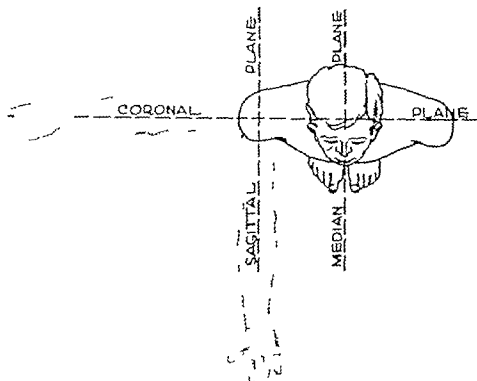


FIG. 22 Planes through or in relation to which the arm moves at the shoulder joint (Codman: *The Shoulder* p. 40)

the components of the shoulder joint mechanism before one can comprehend the complexity of motion at the shoulder joint. That failure to standardize the terminology has led to much confusion is very evident by the literature on movements of the shoulder.

Codman was the first to try to unravel this entanglement. He evolved a most practical group of terms to designate the movements of the shoulder. Moreover, he made many pertinent clinical observations referable to the mechanism of the shoulder which will be recorded in this section.

Terms expressing the position of the distal segment of a joint, such as the knee or the elbow, are not applicable to the shoulder

four joints (scapulohumeral, scapulothoracic, sternoclavicular and acromioclavicular joints) of the shoulder unit participate synchronously.

ANATOMIC POSITION OF THE EXTREMITY

In man, the position of standing with the arms at the side and the palms directed forward is designated the anatomic position (Fig. 23).

ELEVATION

The arm pointing directly upward is designated "elevation." It may be attained by bringing the arm up in the coronal plane.

in the sagittal plane or in a plane midway between the two

ABDUCTION

Elevation in the coronal plane is called abduction. To achieve complete elevation in the coronal plane, the arm must be rotated externally. If the arm is fully rotated internally and then raised in the coronal plane it will lock below the horizontal position. Elevation to the vertical position can be attained only if the arm is first rotated externally 90° . Locking at the horizontal position is brought about by impingement of the tuberosities against the acromion process by external rotation of the arm; the tuberosities are placed so as to pass beneath the acromion when the arm is elevated to 180° .

FORWARD FLEXION

Elevation of the extremity in the sagittal plane is termed "forward flexion." This position is only possible when the arm is rotated internally. Forward flexion in man is the equivalent of extension in the quadruped. From the anatomic position, if the arm is elevated in the sagittal plane, it will lock about 45° above the horizontal. Here further external rotation is nihil by internally rotating the arm 45° it can now be raised to complete elevation.

PIVOTAL POSITION

Regardless of what plane the arm ascends to reach complete elevation the ultimate position of the humeral head in relation to the glenoid cavity, the coracoid process and the acromion process is the same. Codman designated this the pivotal position. In this position no rotation is possible, either internally or externally. A fracture, a dislocation or both will occur if the humerus is forced beyond the fixed position.

With the arm at the side in the anatomic position and the elbow flexed, the condyles of the lower end of the humerus are in the transverse plane. In this position the arm can be rotated internally 90° and externally

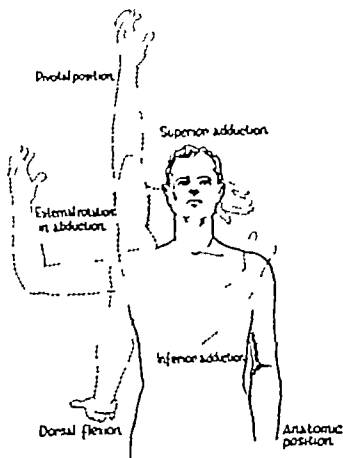


FIG. 23 Different positions of the arm and their designations (Codman The Shoulder p. 41)

approximately 90° , a total range of roughly 180° . As the arm is elevated, regardless of the plane of ascent, the range of rotation gradually diminishes until the arm reaches the pivotal position. Here the internal condyle of the humerus points forward and no rotation is possible.

During elevation, the arm will lock at levels, depending upon the degree of rotation of the humerus. If rotation in the right plane is not hindered, elevation is completed in a smooth rhythmical fashion. However, if rotation of the humerus in the correct plane is obstructed and the arm is forced beyond the locked position, dislocation or fracture of the humeral head results.

It is essential to understand these characteristics of the shoulder mechanism to comprehend the basic principles underlying the mechanism of dislocation and fractures of the head of the humerus.

DORSAL FLEXION

From the anatomic position the arm is brought backward so that the elbow is posterior to the body

ADDUCTION

Superior adduction" is that position in which the elbow is brought toward the median plane above the level of the shoulders

Inferior adduction —the elbow is below the level of the shoulder and is brought toward the median plane.

Adduction is a position midway between superior and inferior adduction

SCAPULOHUMERAL RHYTHM

Elevation of the arm in a smooth continuous rhythmical motion from beginning to end in which the scapulohumeral scapulothoracic sternoclavicular and acromioclavicular joints participate was designated scapulohumeral rhythm by Codman. This uninterrupted smooth motion is of great clinical significance because any alteration in the symmetry of the movement is indicative of some such pathologic disorder as lesions of the musculotendinous cuff, calcareous tendinitis, frozen shoulder, etc.

MOTION AT THE SHOULDER JOINT

The prevailing concepts on movements at the shoulder must be revised in the light of the work of Inman, Saunders and Abbott on the functional mechanism of the shoulder joint. These workers carried still further the observations of Codman. They broke down the movements of the shoulder joint into successive phases and established the sequence and the component of the shoulder joint responsible for each phase. Moreover, they evolved a new fundamental principle in shoulder mechanism, the muscle force couple. This necessitates a revision of the present-day methods or designing new ones to relieve paralytic disorders about the shoulder region. (The observa-

tions made by the above investigators are recorded in the subsequent section.)

The concept is fallacious—that in abduction of the arm the first 90° occurs in the glenohumeral joint and the next 90° in the scapulothoracic articulation (rotation of the scapula on the chest wall). Elevation of the extremity in flexion and abduction is the result of synchronous and continuous motion of all the elements of the shoulder joint complex. Four elements or components constitute this complex: (1) the glenohumeral, (2) the sternoclavicular, (3) the acromioclavicular and (4) the scapulothoracic joints.

GLENOHUMERAL JOINT MOTION

Elevation of the arm in the coronal (abduction) or in the sagittal plane (forward flexion) is produced by simultaneous motion in both the glenohumeral and the scapulothoracic articulations. During the first 30° of abduction and 60° of flexion, the scapula finds a position of stability in relation to the humerus. This is attained either by the scapula's remaining fixed (all motion occurring in the glenohumeral joint until the position of stabilization is reached) or shifting laterally or medially or even oscillating until it is fixed by the humerus. This irregular preliminary period was designated the setting-stage.

From this point the ratio of motion in the two joints is constant until full elevation is complete. The ratio is two humeral and one scapular 10° occurring at the glenohumeral and 5° at the scapulothoracic articulation for every 15° of motion. The total scapular motion is 60° and the glenohumeral 120°. Also it was demonstrated that motion may occur independently in the above joints. By fixation of the scapula the arm can be abducted to 90° passively and 120° actively. Loss of scapular motion however decreases the power of abduction by a third.

The clavicle plays a significant role in elevation of the arm. Any interference with normal motion at either end of the clavicle

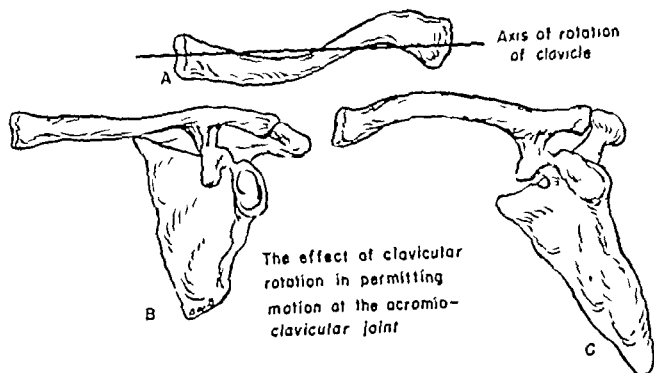


FIG 24 The clavicle rotates on its long axis like a "crankshaft" this is made possible by the configuration of the clavicle whose outer third is curved, producing a relative lengthening of the coracoclavicular ligament when the clavicle rotates on its long axis. (Redrawn from Inman Saunders and Abbott J Bone & Joint Surg 26 2)

is reflected in the total range of elevation. Normal scapular rotation on the chest wall is dependent upon unrestricted motion at the sternoclavicular and the acromioclavicular joints.

STERNOCLAVICULAR JOINT MOTION

This joint permits 40° elevation of the clavicle during elevation of the arm, the ratio being 4° elevation of the clavicle for every 10° elevation of the arm. The range of clavicular elevation is expended during the first 90° elevation of the arm.

ACROMIoclAVICULAR JOINT MOTION

In this joint 20° of motion occurs. Motion occurs in two different phases of elevation: part of it takes place in the first 30° and the remainder after 135° elevation has been attained. The clavicle is bound firmly to the coracoid process and the acromion by the coracoclavicular ligaments. Motion of this joint is made possible only by the configuration of the clavicle.

Its outer one-third is curved so that a relative lengthening of the coracoclavicular ligaments occurs upon rotation on its long axis, thereby allowing a certain range of free motion at this joint.

The clavicle really acts like a "crankshaft" (Fig 24). The amount of motion occurring in both the sternoclavicular and the acromioclavicular joints is equivalent to the amount of scapular rotation on the chest wall (60°).

PRACTICAL SIGNIFICANCE OF ABOVE OBSERVATIONS

It becomes apparent that obliteration of motion at either end of the clavicle surgically or by disease results in restriction of arm elevation equal to the amount of motion lost in the affected joints. Therefore reconstructive surgical procedures should aim to preserve or restore motion in the sternoclavicular and the acromioclavicular articulations. Arthrodesing procedures of these joints or their equivalents are never

justified. On the other hand, excision of the outer end of the clavicle, combined with arthrodesis of the glenohumeral joint, may be a valuable step to increase the range of abduction (Inman, Saunders and Abbott).

MECHANICAL FORCE REQUIREMENTS FOR MOTION AT THE SHOULDER JOINT

The meticulous work of the aforementioned observers disclosed, furthermore, that equilibrium at the glenohumeral joint,

regardless of the position of the arm, was the result of three forces: (1) the weight of the extremity, acting at its center of gravity, (2) muscular masses responsible for abduction (chiefly the deltoid) and (3) the resultant of the above two forces which acts through a center of rotation but in an opposite direction to that of the deltoid.

The third force in turn, is the resultant of two other components: namely, (1) a passive component, the friction and the pressure of humeral head against the

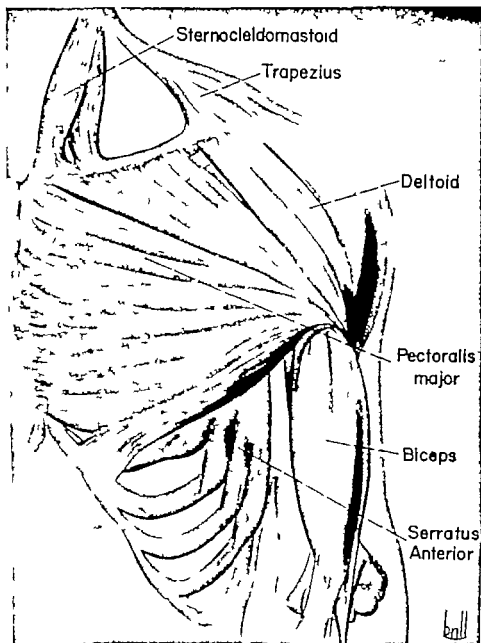


FIG. 25 Outer muscles of the anterior region of the shoulder

glenoid cavity and (2) the active component, the downward pull of the infraspinous muscles (the infraspinatus, the teres minor and the subscapularis muscles). This last force is represented by a force acting at right angles to the plane of the glenoid

cavity and the other parallel to the axillary border of the scapula

The force of elevation (pull of the deltoid) together with the active component of the third force (downward pull of the infraspinous muscles) establishes the mus-

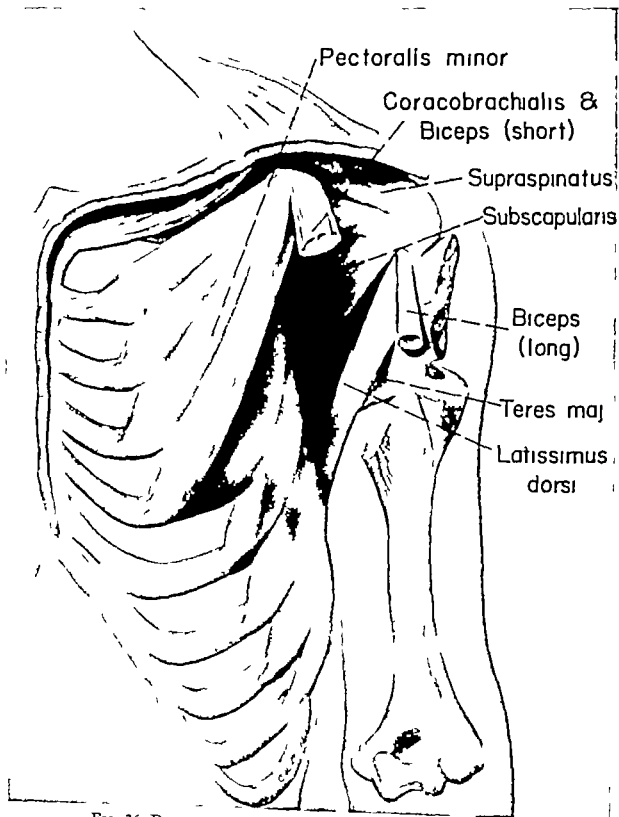


FIG 26 Deep muscles of the anterior region of the shoulder

cle force couple" necessary for elevation of the limb. It was observed that the magnitude of the force required to bring the limb to 90° elevation was at this point, 8.2 times the weight of the extremity. Past 90° the force requirements decreased progressively, reaching zero at 180°. The force requirements of the lower component of the muscle force couple (downward pull of the infraspinous muscles) attained its maximum at 60°, at which point the force requirements were 9.6 times the weight of the limb. Past 90° a progressive decrease of the magnitude of the force was noted reaching zero at 135°.

The same basic principle of the muscle force couple operates in scapular rotation. Three forces are responsible for rotation of the scapula on the chest wall during elevation of the extremity: (1) force which supports the weight of the shoulder girdle operating in a vertical and upward direction; (2) force acting on the acromion in a medial direction; and (3) force acting on the inferior angle of the scapula (serratus anterior muscle).

The force operating from the region of the acromion is a resultant of a passive component represented by the resistance of the clavicle and an active component: the upper portion of the trapezius which here assumes a dual function by aiding in the support of the shoulder girdle.

The second and the third forces establish the muscle force couple essential for rotation of the scapula; they operate in opposite directions and are of equal magnitude reaching their maximum at 90° elevation then dropping progressively to zero at 180°.

Some very significant characteristics are exhibited by the trapezius muscle. At rest its function is entirely supportive during the early phase of elevation of the arm from zero to 35° because of the shift in the angle of action of the muscle; it assumes a dual function: supportive and rotatory. Beyond 35° to 140° its rotatory efficiency progressively increases reaching its maximum at 90°. Beyond 140° its supportive efficiency

steadily increases while its rotatory decreases.

Experiments conducted by Inman, Saunders and Abbott on muscle activity during motion in living subjects have uncovered observations of great significance and importance relative to the mechanism of the shoulder joint. As a result of these studies muscles participating in shoulder motion were grouped into functional units or groups.

ADDUCTOR AND FLEXORS OF THE HUMERUS

This group comprises (1) deltoid, (2) pectoralis major and (3) supraspinatus muscles; all three act as a single functional unit in adduction and flexion of the arm. The sum of the action currents of the individual muscles of this group produces a smooth, regular curve in both flexion and adduction. The curve of flexion reaches its summit at 110°; the curve of adduction at 90°; the former is of slightly greater amplitude.

Deltoid. In adduction the total potential of this muscle is of greater amplitude than that demonstrated in flexion.

Pectoralis Major. Various portions of this muscle exhibit differences in activity depending upon the character of the motion executed. In adduction no activity is demonstrable in any portion of the pectoralis major. In flexion the clavicular portion of the muscle is most active, acting synchronously with the deltoid. Some activity but to a lesser degree is also discernible in the manubrial fibers of the sternocostal head. The remaining fibers of the pectoralis major disclose no cavity in flexion or adduction.

Supraspinatus. The most significant feature of the supraspinatus is that it operates synchronously and simultaneously with the deltoid in all phases of adduction and flexion. Codman made this observation but failed to demonstrate it experimentally as the above workers have so clearly done. This observation dispels the old concept that the supraspinatus muscle initiated ab-

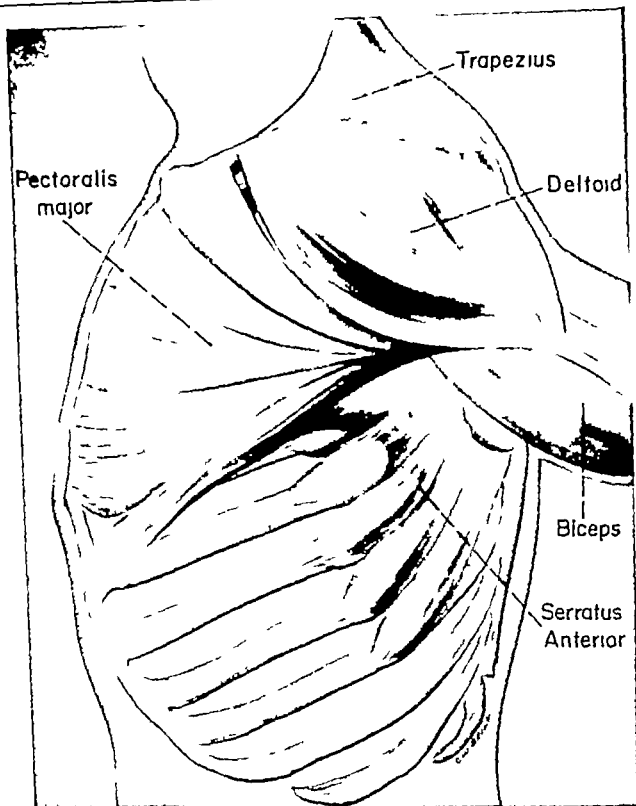


FIG 27 Outer muscles of lateral region of the shoulder

duction and thereafter the movement was completed by the deltoid muscle

DEPRESSORS OF THE HUMERUS

The infraspinous muscles comprise this unit, including the subscapularis the infra spinatus and the teres minor muscles. As

previously indicated, this functional unit constitutes the inferior component of the muscle force couple during flexion and abduction of the extremity. All three elements function simultaneously throughout the entire arcs of motion.

Despite the fact that the teres minor

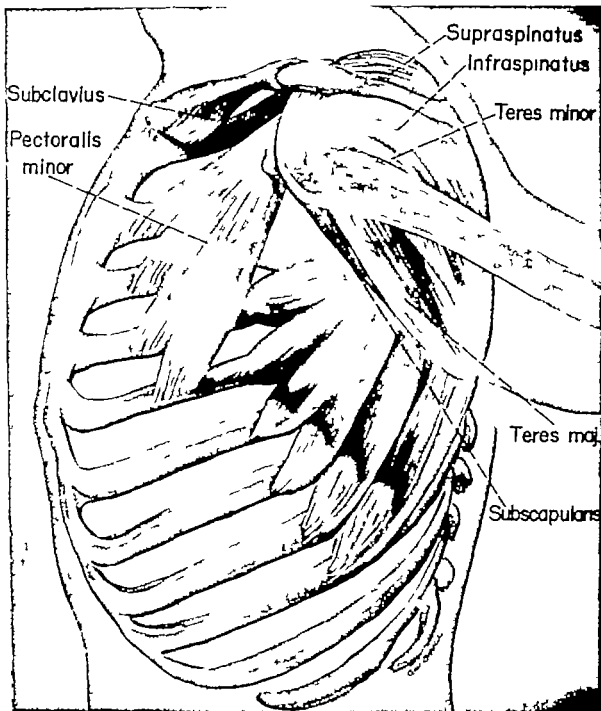


FIG 28 Deep muscles of the lateral region of the shoulder

from a morphologic viewpoint originally was part of the deltoid muscle it behaves in a fashion similar to the infraspinatus throughout its various phases of activity both in flexion and abduction of the extremity

SCAPULAR ROTATORS

This important functional group comprises the trapezius the serratus anterior

and the levator scapulae muscles Various portions of the trapezius and the serratus anterior perform different functions The upper component of the muscle force couple is required for rotation of the scapula and consists of the upper portion of the trapezius the levator scapulae and upper digitations of the serratus anterior All three elements operate as a single unit with two functions besides being the upper compo-

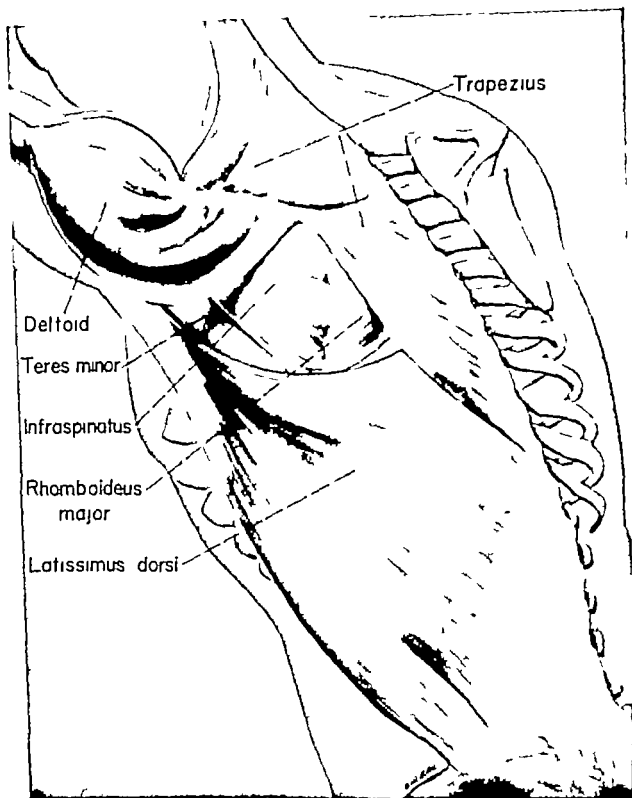


FIG. 29 Outer muscles of the posterolateral region of the shoulder

nent of the force couple necessary for scapular rotation. The unit supports passively the shoulder girdle and also elevates it.

The inferior component of the force couple consists of the lower portions of the trapezius and the lower digitations of the serratus anterior. Both act continuously in a similar fashion except that in abduction

the lower trapezius is more active than in flexion, during which movement it relaxes in order to permit the scapula to slip forward. At this phase of flexion the serratus anterior (lower digitations) assumes the predominant role of the lower force couple.

The middle portion of the trapezius and the rhomboids act as stabilizers of the scap-

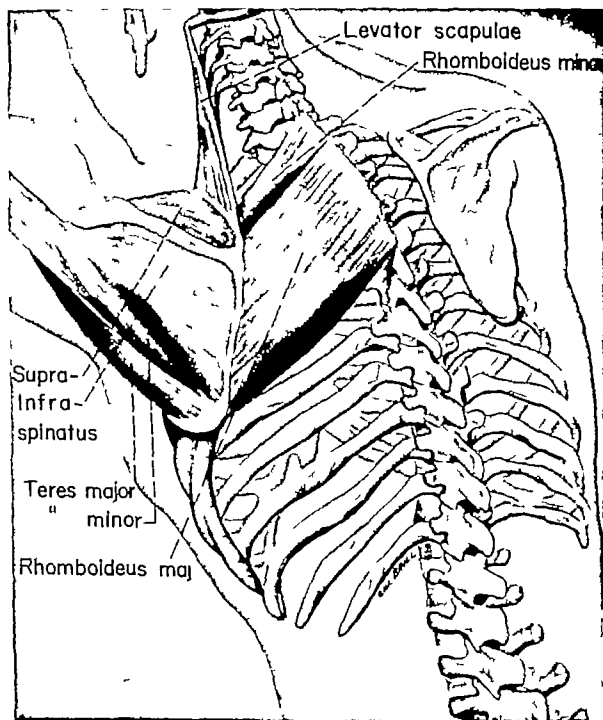


FIG 30 Deep muscles of the posterolateral region of the shoulder

ula during abduction relaxing in flexion to permit rotation of the scapula on the thorax.

It was interesting to note the role of the teres major muscle in the functional mechanics of the shoulder joint. It discloses no evidence of any activity during motion activity is demonstrable only when maintenance of a static position is required

PRACTICAL SIGNIFICANCE OF MUSCLE FORCE COUPLE PRINCIPLE

In the light of the aforementioned observations it is obvious that free complete motion at the shoulder joint can be achieved only when efficient force couples (as de

scribed for glenohumeral motion and scapular rotation) are functioning. Therefore, as pointed out by Inman, Saunders and Abbott, all reconstructive procedures about the shoulder aiming to restore motion in paralytic disorders of this region must re-establish the force couple mechanism which insists on rearrangement rather than substitution of muscles. The belief that power in the deltoid assures abduction of the arm must be discarded.

Regardless of the magnitude of deltoid activity, abduction cannot be achieved without an efficient inferior component of the force couple acting on the humeral head (depressing action of the infraspinatus muscles). This essential component may be restored by transplantation of the latissimus dorsi and the teres major to the postero-inferior surface of the greater tuberosity. If these muscles are paralyzed, some stabilization of the glenohumeral joint may be attained by utilizing the biceps tendon as in the Nicola procedure or one of its modifications.

It has been recorded that only the clavicular head of the pectoralis major muscle functions as an elevator of the arm in flexion; hence, this portion alone can be utilized as an abductor; the remaining portions showing activity only in adduction and medial rotation of the limb. Any procedure designed to use the sternocostal elements as abductors is doomed to failure. No amount of re-education will result in independent contraction of only a portion of the pectoralis major; it always performs as a whole when voluntarily contracted. Therefore, its sternocostal portion can be utilized as a lateral rotator.

According to Inman, Saunders and Abbott, the muscles supporting the scapula (the levator scapulae, the rhomboids and the upper and the middle portions of the trapezius) make it possible for the force couple essential for scapular rotation to operate. In paralysis of these supporting muscles, the scapula may be stabilized by

fascial transplant from the lower cervical spinous process to the base of the spine of the scapula or its upper border (Lowman), thereby re-establishing the fulcrum which is necessary for performance of the force couple.

Muscles such as the teres major which demonstrate activity only in static positions, cannot be made to assume active roles during motion of the extremity. Therefore the teres major cannot be utilized to replace the infraspinatus group.

GLIDING MECHANISM OF BICEPS TENDON

The concept that the biceps tendon moves up and down the groove during motions at the glenohumeral joint must be discarded. With the bicipital tendon and groove exposed under local anesthesia, it can be demonstrated on living subjects that the biceps tendon remains fixed in the groove during motion, but the head of the humerus glides up and down the tendon. Contraction of the biceps muscle by supinating the forearm or flexing the elbow makes the tendon taut but produces no motion of the tendon in the groove. All movements at the shoulder joint regardless of the plane in which the arm is elevated are accompanied by gliding motions of the humerus on the tendon.

Internal rotation of the arm forces the biceps tendon to perform over the medial wall of the groove, the lesser tubercle, from a mechanical viewpoint, functions as a trochlea. Such a position forces the tendon to work at a great mechanical disadvantage. External rotation of the arm places the tendon over the top and the center of the head of the humerus and on the floor of the groove. In this position the mechanical efficiency of the tendon is greatly enhanced and can act as a depressor of the head of the humerus and participate as an abductor of the limb, a function it normally performs in lower forms.

IMPAIRMENT OF MUSCLE FORCE COUPLE BY ISOLATED NERVE PARALYSIS AND OTHER LESIONS

Impairment of the efficiency of the muscle force couples acting upon the humerus and the scapula by isolated muscle paralysis results in varying degrees of disability, depending upon the muscles involved and the importance of these components in the performance of the force couple. Isolated paralysis of the deltoid, the infraspinatus and the serratus anterior muscles causes great disability while paralysis of the

supraspinatus and the trapezius does not seriously impair the functional mechanism of the shoulder.

DELTOID PARALYSIS

Permanent axillary nerve palsy is followed by pronounced impairment of the upper muscle force couple necessary for abduction and flexion of the extremity. The deltoid mass is the most essential component of this force couple. Occasionally a few degrees of abduction may be achieved by the supraspinatus and the clavicular head of the pectoralis major muscles.

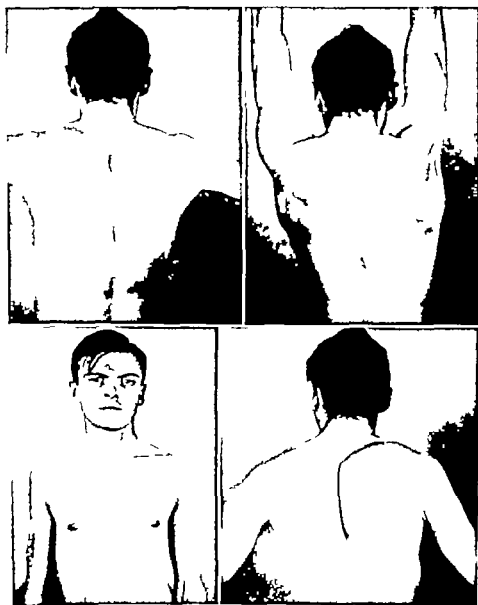


FIG. 31 The ability of an individual with paralysis of the right deltoid muscle to abduct the arm with normal scapulohumeral rhythm and good power

In rare instances, complete abduction is possible in spite of paralysis of the deltoid. Abduction is accomplished by a hypertrophied supraspinatus muscle. One such case has been studied by the author (Fig 31). The patient was able to initiate abduction and carry the movement to completion with normal scapulohumeral rhythm and good power. He participated without difficulty in such athletic activities as baseball and golf. His only complaint was fatigue after an unusually active day. Several such cases have been reported in the literature. Generally, however, the supraspinatus fails to compensate for loss of deltoid power, and the resulting impairment of function is severe.

RUPTURE OF THE SUPRASPINATUS TENDON

Paralysis of the supraspinatus muscle alone is an exceedingly rare lesion, however rupture of the supraspinatus tendon (a relatively common disorder) is its functional equivalent. For a long time following the work of Coleman it was accepted that the supraspinatus muscle occupied a position of great significance in the mechanism of elevation of the extremity. Clinical investigation has re-evaluated the importance of this muscle and has placed it in a position of lesser significance.

Electromyographic studies disclose that the supraspinatus functions simultaneously and continuously with the deltoid muscle and that it is not the initiator of abduction. This observation is confirmed clinically in cases with rupture of the supraspinatus tendon which possess normal scapulohumeral rhythm and abduction power. This point is further emphasized by the investigation conducted on a series of shoulder joints, obtained postmortem from individuals who prior to their death exhibited no clinical disorders of their shoulders (see pp 105-107, Chap 3). Rupture of the supraspinatus tendon was a frequent abnormality in the shoulders of individuals past middle life yet this lesion gave rise to no demonstrable clinical dysfunction of the extremity.

It becomes clear that the role the supraspinatus muscle plays in the efficiency of the upper muscle force couple designed for elevation and flexion of the limb, is a minor one. Moreover, it is evident that the humerus can attain a fulcrum on the glenoid cavity without an intact supraspinatus tendon.

RUPTURE OF THE MUSCULOTENDINOUS CUFF

It will be shown in (pp 105-107) Chapter 3 that the size of the tear (except in cases of complete avulsion of the cuff) is not the factor that determines the degree of functional impairment in the shoulder. The degree of loss of function depends upon the severity of muscle imbalance between the deltoid muscle and the rotator muscles resulting from rupture of the cuff. Extensive tears may produce no severe impairment of function if the remaining portion of the intact cuff is capable of stabilizing the head of the humerus against the glenoid cavity and balancing the power of the deltoid muscle. If a very powerful deltoid muscle exists, as is found in laborers, a relatively small tear may be sufficient to disturb the above muscle balance sufficiently to cause marked impairment of function.

PARALYSIS OF SERRATUS ANTERIOR

The exposed position of the long thoracic nerve makes it vulnerable to trivial injuries, a condition frequently encountered in young athletic individuals. One case in the author's files resulted from direct pressure on the nerve by an osteochondroma located on the ventral surface of the scapula. Fortunately, most acute lesions are not complete, and recovery is the rule.

Complete lesions result in total impairment of the mechanism of scapular rotation. Both the upper and the lower force couples are affected. The trapezius muscle is unable to compensate for the loss of power of the serratus anterior muscle. Abduction is impaired severely and is accompanied by a shift of the scapula toward the



FIG 32 Incomplete paralysis of the serratus anterior muscle resulting from pressure on the long thoracic nerve by an osteochondroma on the ventral surface of the scapula. Note the winging of the scapula. Abduction of the arm was not impaired (7 days after removal of a bone tumor)

vertebral column, there is marked winging of the scapula on motion resulting from failure of the serratus anterior to fix the scapula on the chest wall

In Incomplete lesions, weak abduction is possible, but winging of the scapula is still a prominent feature. Pushing forward against resistances causes pronounced backward displacement of the vertebral border of the scapula, accentuating the winged deformity (Figs. 32 and 33)

PARALYSIS OF TRAPEZIUS

Complete lesions of this muscle also cause serious impairment of the scapular rotatory force couple. The scapula is displaced and rotated downward and outward. Attempts at abduction, which is only possible to the horizontal or slightly below, accentuates the deformity while forward flexion diminishes it (Fig. 34). However, cases have been reported in which lesions of the trapezius did not disturb the normal mechanism of the shoulder to any appreciable degree motion



FIG 33 Stability of the scapula restored after recovery of the serratus anterior muscle (6 weeks after operation)

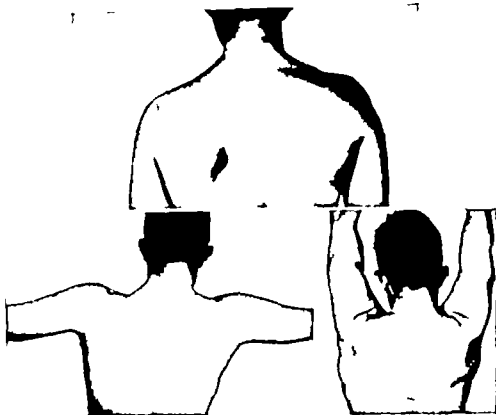


FIG 34 Paralysis of left trapezius muscle, caused by penetrating wound of the right side of the neck. (*Top*) Lateral and downward displacement of scapula and flaring of its vertebral border. (*Left*) Maximum range of abduction (90°) reveals return of power to the muscle after spontaneous regeneration of the accessory nerve. (*Right*) Complete abduction of the arm is now possible. Time of regeneration was 11 months (Haymaker and Woodhall *Peripheral Nerve Injuries*, Philadelphia, Saunders)

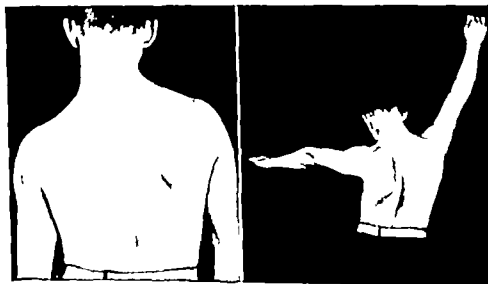


FIG 35 Complete paralysis of left trapezius and serratus anterior muscles caused by penetrating wound implicating the accessory and the long thoracic nerves. At rest the left shoulder droops, and slight flaring of the vertebral border of the scapula is discernible. Elevation is possible to 90° only in the forward plane. Observe the pronounced flaring of the inferior angle of the scapula when the above position of elevation is maintained. (Haymaker and Woodhall *Peripheral Nerve Injuries* Philadelphia Saunders)



FIG 36 Age, 2 months no centers of ossification for the head of the humerus are demonstrable.

in flexion and abduction was rhythmical and powerful. Apparently the serratus anterior, in certain instances, can compensate to assume the work of the trapezius in both the upper and the lower force couples.

Paralysis of both the trapezius and the serratus anterior results in pronounced dysfunction of the shoulder mechanism. Attempts at elevation produce a very characteristic deformity. Some abduction of the humerus occurs at the scapula, but the scapula fails to rotate outward and upward. Instead, it rotates downward. Motion is accompanied by marked winging of the vertebral border and inferior angle of the scapula (Fig 35).

OSSIFICATION OF THE UPPER END OF THE HUMERUS

At birth a small ovoid nucleus for the head of the humerus may be present; more frequently this center of ossification does not appear until the fourth or the sixth month (Fig 36). During the second year of life a nucleus for the greater tuberosity ap-



FIG 37 (*Left*) Age 14 months a large nucleus for the head and a smaller one for the greater tuberosity is plainly visible.

FIG 38 (*Right*) Same patient at 2 years of age.



FIG. 39 (*Left*) Age 4 to 5 years note the appearance of a third epiphysis which is destined to be the lesser tuberosity
FIG. 40 (*Right*) Age 7 years, greater and lesser tuberosities have fused into one mass. This usually occurs during the fifth year

pears lateral to that for the head (Fig. 37 and 38) The epiphysis for the lesser tuberosity appears during the fifth year (Fig. 39) it is rarely possible to demonstrate all three centers of ossification on one roentgenogram The nuclei for the tuberosities fuse into one mass in the fifth year (Fig. 40) which in turn unites with that of the head of the humerus between the twelfth and the fourteenth years (Fig. 41) The head unites with the shaft of the humerus during the nineteenth year

CONGENITAL ABNORMALITIES

CONGENITAL HIGH SCAPULA (SPRENGEL'S DEFORMITY)

This anomaly also referred to as undescended scapula and elevated scapula, is by far the most frequently encountered congenital deformity of the shoulder girdle. In this condition the scapula which arises as a cervical appendage, fails to migrate cau-



FIG. 41 Age 12 Epiphysis of the tuberosities has united with that of the head. Observe a small nucleus at the end of the coracoid process and one for the end of the acromion. The latter appears between 12 and 15 years and usually comprises three or more centers of ossification.



FIG 42 (Left) Age, 17 and 18 years. The epiphyseal plate between the head and the shaft of the humerus is markedly thinned as is the one at the end of the acromion.

FIG 43 (Right) Age, 19. Fusion between the head and the shaft of the humerus is completed.

dally, during early fetal development to its normal position on the thorax

Normally the scapula meets the upper portion of the thorax by the end of the third month. Occasionally however the scapula

fails to free itself from the cervical vertebrae and remains attached to the cervical spine by its vertebral border or by a fibrous or bony structure varying in size from a thin strip to a large bony plate the supra-capula. This extends from the angle formed by the superior and the inferior vertebral borders to the lower cervical or first thoracic spine. The scapula may be normal in size and shape or smaller than normal with an altered configuration the breadth being increased and the length decreased. Occasionally the deformity is bilateral. The portion above the spine may be angulated sharply forward to conform to the contour of the upper portions of the thoracic cage.

Elevation of the scapula usually is combined with such other congenital anomalies as failure of fusion in the midline of the laminae of some cervical vertebrae, cranial bifidum defects in the upper dorsal vertebrae and with such developmental anomalies of the thoracic outlet as cervical ribs and abnormal first thoracic ribs, hemivertebrae, congenital scoliosis, fusion of ribs and irregular vertebral segmentation. Developmental defects of the shoulder mus-



FIG 44 Sprengel's shoulder in a girl 8 years of age.

culature may exist. All or part of the trapezius may be absent, and the levator scapulae and the rhomboid muscles may exist as strips of fibrous or cartilaginous tissue running from the base of the occiput or pine to the vertebral border of the scapula. Torticollis may be a concomitant deformity.

When the arm is elevated the scapula fails to shift laterally; its lower angle does not rotate outward when the extremity is brought above the horizontal. Little or no dysfunction of the shoulder exists in most instances. While slight restriction of elevation of the extremity in the coronal and sagittal plane usually exists, other movements are not affected. The overall functional efficiency of the shoulder girdle is good.

The undesirable cosmetic appearance of the shoulders rather than dysfunction forces parents to seek medical advice. Surgery, in most instances, does not yield good results, and one wonders whether the operations performed to correct this deformity are justifiable in the face of so many disappointing results. Nevertheless, operation may improve slightly the appearance of the patient and increase the range of motion of the scapula on the thorax; this is particularly true if performed in the early years of life (rarely before the age of 3) before marked adaptative structural changes have occurred which preclude any correction or increase in function of the extremity.

Operative Technique. The operation most commonly employed to correct congenital high scapula is that of Schrock, or a modification of this procedure. Essentially it comprises subperiosteal stripping of the entire scapula displacing it distally as far as possible and fixing it in its new site by suturing its inferior angle to a rib.

A long slightly curved skin incision is made parallel to and one inch to the outer side of the vertebral border of the scapula, extending from above the superior angle to below the inferior angle. All muscle attachments are divided at the vertebral border



FIG 45 (Top) Congenital elevation of the scapula. Observe that the scapula is placed high on the thoracic cage; its breadth is increased and its length decreased. It is rotated outward and forward on its vertical axis and it possesses a suprascapular bone. Note the congenital malformation of the upper thoracic cage and the presence of long cervical ribs. (H Ostrum, Philadelphia General Hospital)

FIG 46 (Bottom) Congenital elevation of the scapula with an omovertebral bone. (H Ostrum)

and detached subperiosteally. The subperiosteal dissection is continued along the ventral surface of the scapula; the suprascapular and the infrascapular surfaces are spined (detaching the trapezius) and the upper border of the scapula. Care is exer-



FIG 42 (*Left*) Age 17 and 18 years. The epiphyseal plate between the head and the shaft of the humerus is markedly thinned, as is the one at the end of the acromion.

FIG 43 (*Right*) Age, 19. Fusion between the head and the shaft of the humerus is completed.

dally, during early fetal development to its normal position on the thorax.

Normally, the scapula meets the upper portion of the thorax by the end of the third month. Occasionally, however, the scapula

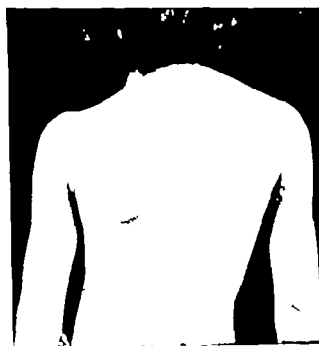


FIG 44 Sprengel's shoulder in a girl 8 years of age.

fails to free itself from the cervical vertebrae and remains attached to the cervical spine by its vertebral border or by a fibrous or bony structure, varying in size from a thin strip to a large bony plate, the supra scapula. This extends from the angle formed by the superior and the inferior vertebral borders to the lower cervical or first thoracic spine. The scapula may be normal in size and shape or smaller than normal with an altered configuration, the breadth being increased and the length decreased. Occasionally the deformity is bilateral. The portion above the spine may be angulated sharply forward to conform to the contour of the upper portions of the thoracic cage.

Elevation of the scapula usually is combined with such other congenital anomalies as failure of fusion in the midline of the laminae of some cervical vertebrae, cranium bifidum, defects in the upper dorsal vertebrae, and with such developmental anomalies of the thoracic outlet as cervical ribs and abnormal first thoracic ribs, hemivertebrae, congenital scoliosis, fusion of ribs, and irregular vertebral segmentation. Developmental defects of the shoulder mus-

culature may exist. All or part of the trapezius may be absent, and the levator scapulae and the rhomboid muscles may exist as strips of fibrous or cartilaginous tissue running from the base of the occiput or spine to the vertebral border of the scapula. Torticollis may be a concomitant deformity.

When the arm is elevated the scapula fails to shift laterally; its lower angle does not rotate outward when the extremity is brought above the horizontal. Little or no dysfunction of the shoulder exists in most instances. While slight restriction of elevation of the extremity in the coronal and sagittal plane usually exists, other movements are not affected. The overall functional efficiency of the shoulder girdle is good.

The undesirable cosmetic appearance of the shoulders rather than dysfunction forces parents to seek medical advice. Surgery, in most instances, does not yield good results, and one wonders whether the operations performed to correct this deformity are justifiable in the face of so many disappointing results. Nevertheless, operation may improve slightly the appearance of the patient and increase the range of motion of the scapula on the thorax; this is particularly true if performed in the early years of life (rarely before the age of 3) before marked adaptative structural changes have occurred, which preclude any correction or increase in function of the extremity.

Operative Technique. The operation most commonly employed to correct congenital high scapula is that of Schrock, or a modification of this procedure. Essentially, it comprises subperiosteal stripping of the entire scapula, displacing it distally as far as possible and fixing it in its new site by suturing its inferior angle to a rib.

A long slightly curved skin incision is made parallel to and one inch to the outer side of the vertebral border of the scapula, extending from above the superior angle to below the inferior angle. All muscle attachments are divided at the vertebral border



FIG. 45 (Top) Congenital elevation of the scapula. Observe that the scapula is placed high on the thoracic cage; its breadth is increased and its length decreased. It is rotated outward and forward on its vertical axis, and it possesses a suprascapular bone. Note the congenital malformation of the upper thoracic cage and the presence of long cervical ribs. (H. Ostrum, Philadelphia General Hospital.)

FIG. 46 (Bottom) Congenital elevation of the scapula with an omovertebral bone. (H. Ostrum.)

and detached subperiosteally. The subperiosteal dissection is continued along the ventral surface of the scapula, the suprascapular and the infrascapular surfaces, the spine (detaching the trapezius) and the upper border of the scapula. Care is exer-

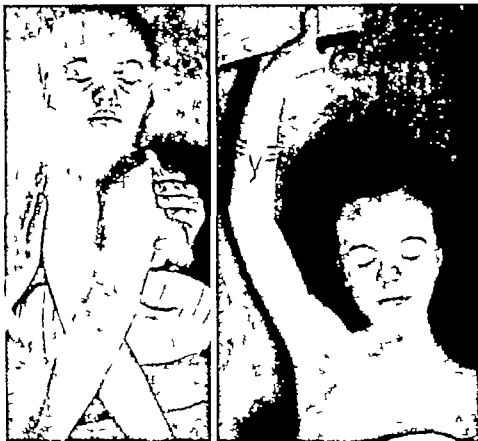


FIG 47 Cleidocranial dysostosis. The shoulders are readily approximated in the midline beneath the chin. Shoulder function is not impaired. (Mercer Orthopedic Surgery London Arnold)

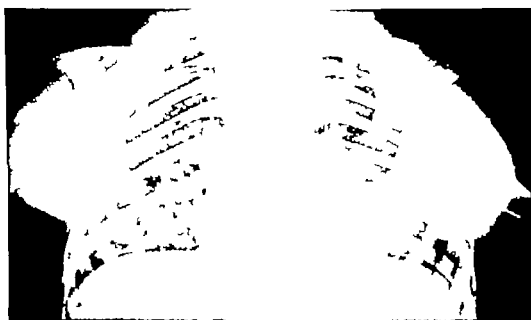


FIG 48 Roentgenogram of a case of cleidocranial dysostosis. Absence of clavicles (H Ostrum)

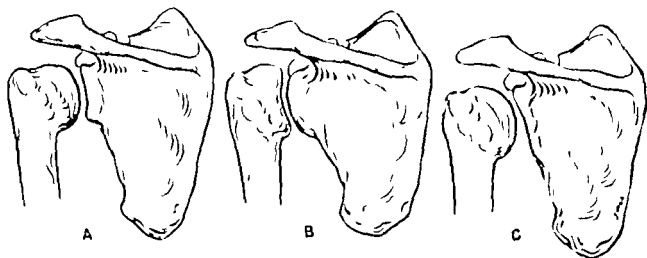


FIG. 49 Congenital deformities of scapulohumeral joint (A) Humerus varus (B) Articular surface of the glenoid cavity is convex and that of the humerus concave (C) Shallow flattened glenoid fossa

cised to avoid injuring the supraclavicular nerve and the transverse scapular artery as they traverse the suprascapular notch. Any fibrous band or osseous bar between the scapula and the vertebral column is excised. The prominent upper portion of the scapula may be removed. The entire scapula is then displaced caudally as far as possible, and the inferior angle is sutured with stout chromic catgut to a rib to ensure maintenance of its new position.

After wound closure the arm is immobilized by a Velpeau dressing or a plaster of Paris swathe (Fig. 198) for 4 weeks, this is followed by physiotherapy to restore normal shoulder function. If adequate displacement of the scapula cannot be achieved by simple subperiosteal detachment of all soft tissue, additional mobilization may be attained by an osteotomy at the base of the acromion.

CLEIDOCRANIAL DYSOSTOSIS

This is an exceedingly rare congenital abnormality and hereditary transmission exists in most instances. Occasionally, however, hereditary plays no part. The characteristic features are:

- 1 Aplasia of the clavicles
- 2 Delay in closure of the cranial suture
- 3 Excessive enlargement of the transverse diameter of the cranium while the

base of the skull and the face remains relatively small.

Cases have been reported in which other congenital defects existed together with such conditions as achondroplasia, defective development of the small bones of the hands and the feet, malformation of the vertebral column, the pelvis and the femora and disturbance of normal dentition. Bones of both membranous and endochondral derivation may be affected.

Varying degrees of aplasia of the clavicles may exist, caused by developmental failures of only a small portion (usually the acromial end) to absence of the entire clavicle. There may also be abnormal development of the large shoulder girdle muscles, particularly the pectoralis major, the deltoid and the trapezius. The clavicular portion of the trapezius and the deltoid muscles may be deficient or absent.

As a rule the condition causes no shoulder dysfunction. The excessive mobility of the girdles does not impair the efficiency of the extremity. Individuals so afflicted can readily bring together the tips of their shoulders in the midline beneath their chins. Rarely is any form of therapy indicated. Occasionally, however, one of the free ends of the clavicle presses on the subclavicular structures and in such instances resection of the part is justified.

HUMERUS VARUS

The angle of the head of the humerus is less during fetal development than that existing in the adult state. But occasionally this fetal type relationship of the head to the shaft of the humerus is found in adults and is designated humerus varus.

In the normal humerus the head is directed upward, making an angle of 180° to 140° with the shaft whereas, in humerus varus the head is directed sidewise at an angle of 100° or less. Usually the affection is bilateral.

There is disagreement as to the true cause of this deformity. Some workers contend that it is a developmental defect but maintain also that it may be the result of stress applied to soft demineralized bone during the acute phases of such diseases as rickets, scurvy and hypothyroidism producing a varus deformity of the humerus similar to that of coxa vara. Cases are on record in which the defect was observed in normal individuals.

OTHER CONGENITAL DEFORMITIES OF THE GLENOHUMERAL JOINT

Two other congenital abnormalities of the glenohumeral joint are encountered occasionally. In one the glenoid cavity appears as a convex protuberance which articulates with a concave articular surface on the medial aspect of the head of the humerus (Fig. 49B). In the other the glenoid fossa is flattened. Brailsford points out that radiographically it appears as if the epiphysis of the lower rim of the glenoid cavity has failed to develop (Fig. 49C); the defect is bilateral.

CORACOCALVICULAR JOINT

Another rare bilateral developmental abnormality is the appearance of a bony process on the inferior aspect of the outer

one-third of the clavicle which articulates with a similar process arising from the coracoid process. This mechanism replaces the coracoclavicular ligament (the conoid and the trapezoid ligaments). Cases have been reported in which complete ossification of the aforementioned ligaments occurred; trauma was the initiating factor in most of them.

CONGENITAL DISLOCATION OF THE SHOULDER

While such an entity has been definitely established, true congenital dislocation of the shoulder is rarely encountered. Undoubtedly many of the reported cases were instances of dislocation sustained at birth or after birth subsequent to some form of obstetric paralysis.

Dislocation occurring at birth is denied by some authorities (Sever, Codman, Taylor). Others are firmly convinced that it occurs at the time of birth and that obstetric palsy is really a pseudoparalysis produced by secondary involvement of the brachial plexus by organizing inflammatory tissue in the axilla (T. T. Thomas).

The most comprehensive study was made by Grieg (1924) in which the majority of the reported cases were posterior dislocations with a few anterior or inferior dislocations. In the posterior type the glenoid was not palpable; the head of the humerus occupied a posterior position usually beneath the spine; the arm was rotated internally and the forearm was flexed slightly. All movements were restricted particularly elevation and external rotation. Both the acromion and the coracoid were elongated and bent forward in front of the head of the humerus. No evidence of paralysis of any muscle group exists in these cases. Treatment is similar to that described for obstetric palsies.

BIBLIOGRAPHY

- Beever C E. The Cronian lectures on muscular movements and their representation in the central nervous system. Lecture II. Brit. M. J. 1 141, 1421 1903.
- Bowen, W. P. Applied Anatomy and Kinesiology: the Mechanism of Muscular Movement. ed. 4. Lea & Febiger Philadelphia 1928. p. 16.
- Brunstrom S. Muscle testing around the shoulder girdle: a study of the function of shoulder blade fixators in seventeen cases of shoulder paralysis. J. Bone & Joint Surg. 23 263-272 1941.
- Caldwell, G. D. Treatment of complete permanent acromioclavicular dislocation by surgical arthrodesis. J. Bone & Joint Surg. 25 368 1943.
- Catbcart C W. Movements of shoulder girdle involved in those of the arm on the trunk. J. Anat. (Paris) 18 211 218 1884.
- Codman, E. A. The Shoulder. Boston: Thomas Todd Co. 1934.
- Critchley M. Sprengel's deformity with paralysis. Brit. J. of Surg. 14 243 1927.
- Cyrax, E. F. A case of cleido-cranial dysostosis. Edin. Med. J. 30 600 1923.
- Davidson W. D. Traumatic deltoid paralysis treated by transplantation. J.A.M.A. 106 2237 1936.
- Duchenne G. B. Physiologie des mouvements. Paris: Baillière 1867. (tr. E. B. Kaplan Philadelphia: Lippincott 1949).
- Elmaleh R. C. Proc. Roy. Soc. Med. Ortho., No. 8 18 25 1925.
- Fitchet, S. M. Cleido-cranial dysostosis hereditary and familial. J. Bone & Joint Surg. 11 838 866 1929.
- Gillies H. D. Proc. Roy. Soc. Med., p. 1003 1934.
- Grant. An Atlas of Anatomy. ed. 2. Baltimore: Williams & Wilkins 1947.
- Greig D. M. Edin. Med. J. 16 49 1916.
- Greig D. M. Congenital High Scapula. Edin. Med. J. 31 22 1924.
- Haas, S. L. The treatment of permanent paralysis of the deltoid muscle. J.A.M.A. 104 99 1935.
- Haymaker W., and Woodhall B. Peripheral Nerve Injuries. Philadelphia: Saunders 1945.
- Heineke. Ztschr. f. Orthop. Chir. 21 553 1908.
- Inman V. T. Saunders J. B. deC. M. and Albott L. C. Observations on the shoulder joint. J. Bone & Joint Surg. 26 130 1944.
- Jackson B. H. Undescended scapula with an omio-vertebral bone. Radiol. 19 67 1932.
- Klippel et Feil. Anomalie de la colonne vertebrale. Bull. et Mem. Soc. Anat. de Paris 87 185 1912.
- Lochari R. D. Movements of the normal shoulder joint and a case with trapezius paralysis studied by radiogram and experiment in the living. J. Anat. 64 283-302 1930.
- McBride E. D. Congenital deficiency of the clavicle. J. Bone & Joint Surg. 9 545 1927.
- Martin C. P. Movements of the shoulder joint with special reference to rupture of the supraspinatus tendon. Am. J. Anat. 66 213 234 1940.
- Mayer L. Transplantation of the trapezius for paralysis of the abductors of the arm. J. Bone & Joint Surg. 9 412 1927.
- Middleton D. S. Studies of striated muscles as a cause of congenital deformity, Edin. Med. J., p. 401 1934.
- Milgram J. E. Shoulder Anatomy. Regional Orthopaedic Surgery and Fundamental Orthopaedic Problems. J. W. Edwards Ann Arbor 1947.
- Moseley H. F. An unusual case of actinomycosis. J. Bone & Joint Surg. 23 359-66 1941.
- Nicotra A. Arch. d. Radiol. 5 194 1929 and 6 19 1930. Radiol. Med., 16 97 1929.
- Persol V. M. Human Anatomy. Philadelphia, Lippincott 1930.
- Pollack, L. J. Accessory muscle movements in deltoid paralysis. J.A.M.A. 89 526 1922.
- Pollack L. J., and Davis L. Peripheral Nerve Injuries. New York: Hoeber 1933. pp. 373-387.
- Seifert E. Fort. d. g.d. Ront. 43 620 1931.
- Staples O. S. and Watkins A. L. Full active abduction in traumatic paralysis of the deltoid. J. Bone & Joint Surg. 25 85 89 1943.
- Stevens J. H. The action of the short rotators on the normal abduction of the arm, with a consideration of their action in some cases of subacromial bursitis and allied conditions. Am. J. M. Sc. 138 870-884 1909.

3

Variational Anatomy and Degenerative Lesions of the Shoulder Joint

GLENOID SIDE OF THE GLENOHUMERAL JOINT
BURSAL SIDE OF THE GLENOHUMERAL JOINT

LESIONS OF THE SHOULDER COMPATIBLE WITH
GOOD FUNCTION

INTRODUCTION

Hitherto an orderly study of the variational anatomy and degenerative lesions of the shoulder had not been done. Although much information on degenerative alterations of this region may be found in the literature, it never has been correlated with a clinical investigation. The study recorded in this chapter was divided into two parts.

In the first part of this chapter the detailed variational anatomy of the inside of the glenohumeral joint was studied and the degenerative abnormalities in all the component elements of the shoulder joint were noted. Special attention was directed to the changes in the glenoid cavity, labrum glenoidale, glenohumeral ligaments, bursal recesses, synovialis, tendon of the long head of the biceps brachii muscle, and the musculotendinous cuff. This phase of the investigation disclosed some very significant observations. It became apparent that one unfamiliar with the variational anatomy of this region might readily misinterpret the findings as pathologic or traumatic disorders. Also, the accumulated data failed to support the rationale of some of the popular operative procedures for stabilization of recurrent dislocation of the shoulder, particularly the suspension methods, and provides an explanation for the success of others, especially those designed to limit external rotation of the extremity, such as the

Bankart and Putti-Platt operation. However, this study points out that many of the successful procedures are based on false premises.

In the discussion on lesions of the shoulder compatible with good function, an attempt was made to evaluate the clinical significance of the observations noted in the first part. It soon became evident that degenerative lesions of varying severity were compatible with good shoulder function and that our present-day concept of a normal shoulder joint needs revision. Furthermore, this investigation disclosed specific degenerative alterations, characterized by a diffuse and progressive process of fibrosis involving all the soft tissue structures of the joint. The progressive nature of this abnormality was revealed by the gradual increase in gradient of this lesion in specimens of successive decades, especially after the fourth. Shoulder joints of the eighth and ninth decades exhibited the most severe changes. As will be shown subsequently, the aforementioned degenerative change is responsible for a progressive decrease in the range of external rotation of the arm in normal individuals, a feature which cures spontaneously recurrent dislocations of the shoulder and for practical purposes eliminates the disease after the fourth decade. On the other hand, the same disorder coupled with other factors appears

to be the responsible causative agent for frozen shoulder

A total of 452 shoulder joints were studied. Two hundred eight of these were obtained from cadavers ranging in age from 14 to 87 years. Of these, 12 were fetuses 6 to 9 months old. No clinical histories were available on these specimens. One hundred forty-four shoulders were obtained postmortem. A history was obtained, and an examination of the shoulders was made on the individuals who provided these specimens, prior to their death. This data was obtained in the following manner: All ward patients on the hospital critical list were questioned concerning any joint disorders and their shoulders were examined. In case of death the glenohumeral joints with the cuffs intact were removed, dissected and studied. Only those joints which failed to give any evidence of disease through history or physical examination were selected for this investigation. Furthermore, this group of specimens furnished the material for microscopic study. In addition to the above specimens, 100 scapulae, devoid of all soft tissues, were utilized for detailed macroscopic investigation of the glenoid cavity.

The first 96 specimens obtained from cadavers were utilized for the macroscopic phase of the investigation. After they were mounted and photographed (Fig. 50) they were arranged in chronologic order to correlate better the findings from one decade to another. From the first 44 postmortem specimens, shoulder joints were selected which were considered representative joints for different decades and whose pathologic alterations corresponded to the representative joints for each decennium used for the macroscopic study. From these representative joints sections were obtained for microscopic study through all the synovial areas: labrum glenoidale, biceps tendon, selected areas of the musculotendinous cuff and from the glenoid cavity. The findings were also recorded chronologically and in tabular form.

For the purpose of study and presentation the glenoid cavity, which is shaped like an inverted comma, was divided into two fields: (1) the upper field, designated the tail of the comma, and (2) the lower field, the head of the comma. These two areas are separated by a transverse line which roughly corresponds to the epiphyseal line of the

GLENOID SIDE OF THE GLENOHUMERAL JOINT

MATERIALS AND METHODS

Materials Used in Study

Glenoid Side of Scapulohumeral Joint

196 shoulders from cadavers—ranging in age from 14 to 87 years	{ No clinical histories or physical examinations were available on these individuals
12 fetuses—ranging in age from 6 to 9 months	
144 shoulders obtained postmortem	{ Clinical history and examination of shoulders obtained prior to death

Bursal Side of Scapulohumeral Joint

100 shoulders	{ No clinical histories or physical examinations were available on these individuals
---------------	--

In addition to the above 100 scapulae devoid of soft tissue were utilized in study on the glenoid cavity.

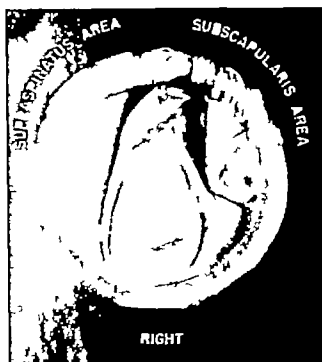


FIG 50 Finished specimen mounted, showing the inside of the glenohumeral joint. Note that the labrum glenoidale surrounding the lower half of the glenoid cavity is continuous with the capsule, while that portion surrounding the upper half is free. Also note that the synovial membrane lining the intracapsular structures (the labrum glenoidale the biceps tendon and the glenohumeral ligaments) is closely adherent to the above structures. In this specimen the superior glenohumeral ligament is a well-defined and distinct structure. The capsule in the anterior aspect of the shoulder joint is not continuous with the labrum it extends toward the base of the coracoid process from which point it is reflected along the neck of the scapula to the capsular surface of the labrum thereby forming a large synovial recess or pouch

glenoid cavity (Fig 51). Likewise the synovial membrane was divided into five areas designated (1) the subscapularis area (2) the area of the coracohumeral ligament (3) the supraspinatus area (4) the infraspinatus and teres minor area and (5) the triceps area (Fig 52).

Synovial pouches found in the anterior aspect of the shoulder joint (often referred to as bursae in textbooks) were found to be related intimately to the middle gleno-

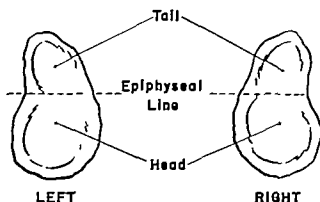


FIG 51 The glenohumeral cavity is shaped like an inverted comma. The upper portion was designated the tail of the comma the lower portion the head of the comma. The transverse line between these two regions roughly corresponds to the epiphyseal line of the glenoid cavity.

humeral ligament and tendon of the subscapularis muscle. Hence they were designated in accordance with the position occupied relative to these structures. The synovial recess above the middle ligament was designated the superior subscapularis recess. The recess below the ligament was designated the inferior subscapularis recess.

The articular cartilage of the glenoid cavity the glenohumeral ligaments the labrum glenoidale the biceps tendon and the fibrotendinous capsule were studied separately. The degree of the gross alterations noted in all components of each shoulder joint was tabulated in chronologic sequence. Gross changes in the articular cartilage of the glenoid cavity were graded from 1 plus to 4 plus. One plus expressed minimal abnormalities of the articular cartilage as mild surface irregularity or a velvetlike appearance of the superficial cartilage. Severe forms of these alterations plus actual splitting fraying and shallow indentations of the articular cartilage were labeled 2 plus. More marked changes plus an articular surface which exhibited irregular extensive areas of erosion were designated 3 plus. Four plus expressed the maximum degree of degenerative changes encountered in the articulating surface. This included advanced fraying flaking, splitting and erosion of the

cartilage, exposed dense eburnated areas of subchondral bone, and new bone formation

Degenerative changes in the labrum glenoidale and tendon of the long head of the biceps brachii muscle consisted of varying

rim of the glenoid cavity in normal shoulder joints. Microscopic examination of the cartilage of the glenoid cavity in infant shoulders revealed that it consisted of hyaline cartilage which was thinner in the center of

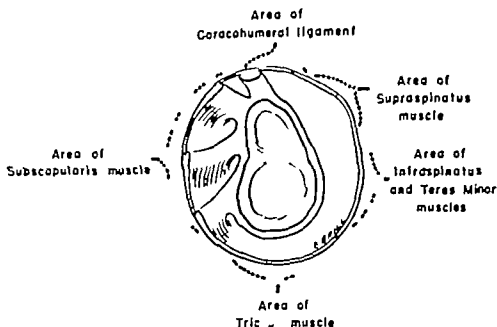


FIG. 52 The various designated fields of the synovial membrane

degrees of shredding, thinning, thickening, areas of nonattachment of the labrum, and occasionally of partial or complete absence of these structures. Changes in the synovial membrane comprised villous formation, synovial tabs and fringes, also thickening due to fibrosis in the subsynovial tissues. Likewise, all of these alterations were graded from 1 plus to 4 plus based on the relative severity of the degenerative changes. Abnormalities in the fibrotendinous cuff, demonstrated by such alterations as fraying, shredding and tearing of the structure, were likewise evaluated on the extent and the severity of the lesions.

OBSERVATIONS REFERABLE TO THE GLENOID CAVITY

The articular surface of the glenoid is a shallow cavity slightly deeper in the region of the head of the comma than in the tail. Its depth is augmented by a triangular fibrocartilaginous structure, the labrum glenoidale, which is attached firmly to the

the head of the comma than in the surrounding areas and the hyaline cartilage in the tail of the comma.

The first observation of any significance referable to the articular cartilage was noted in the second decennium. No macroscopic changes were demonstrable in the tail of the comma. In the center of the head of the comma a distinct circular area was noted in 4 of the 6 specimens belonging to this decade (Fig. 53). It was grossly visible in all specimens for the third and the fourth decades, in 11 out of 12 for the fifth decade, in 15 out of 18 for the sixth decade, in 29 out of 30 for the seventh decade, and in 22 out of 24 for the eighth and the ninth decades.

The increased visibility of the area is dependent upon the fact that here the hyaline cartilage is thinner than the surrounding cartilage. Also, microscopic examination revealed that the earliest and most severe alterations of the cartilage were found in this area. These changes increased in sever-

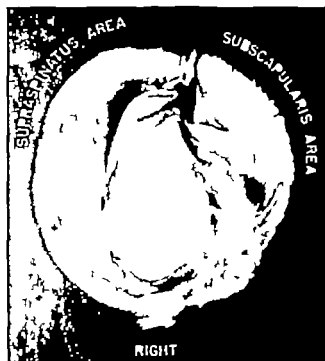


FIG. 53 A circular area is barely visible in the center of the head of the comma. This is a specimen of the second decade. In the subscapularis and the coracohumeral areas of the synovial membrane are fingerlike synovial villi projecting into the lumen of the joint cavity



FIG. 54 Typical hyaline cartilage found in the center of the head of the comma in shoulders of infants. (Photomicrograph $\times 67$)



FIG. 55 Degenerative changes in the hyaline cartilage in the center of the head of the comma, with formation of connective tissue which assumes the characteristics of imperfectly formed fibrocartilage. This is a specimen of the fifth decade. ($\times 136$)

ity in each successive decade leading one to believe that this circular area might be one of greater contact than the surrounding articular cartilage. According to Fick, Poirier was the first to make this observation. However he believed this region of the comma's head to be covered with fibrocartilage. The findings in this study do not substantiate this observation. Microscopic section through the area in question of infant shoulders revealed that hyaline cartilage was the articular tissue found in this region (Fig. 54). In specimens of later decades the severe degenerative alterations of the cartilage in the center of the comma's head and metaplasia of the degenerated hyaline cartilage to imperfectly formed fibrocartilage explain how such an erroneous interpretation may be made (Fig. 55).

Macroscopically alterations were noted as stated in the articular cartilage of the glenoid cavity as early as the second decade. The bluish white glistening resilient, delicate articular cartilage found in infancy gave way to a firm compact opaque tissue demonstrable as early as the second age

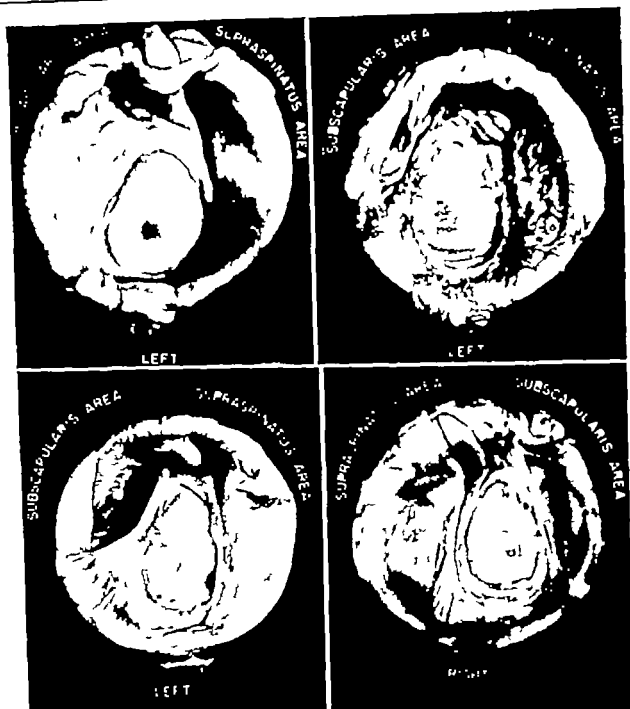


FIG 56 (Top left) Papillary velvet like appearance of hyaline cartilage in the head of the comma, due to degenerative changes. Also note that a portion of the biceps tendon is still extrasynovial lying in a tunnel within the fibrous capsule. Circular area in this specimen is well defined. (Top right) Maximum degenerative changes in the hyaline cartilage of the glenohumeral cavity, more pronounced in the head of the comma. Note that the intracapsular portion of the biceps tendon is absent, and only a small stub remains attached to the supraglenoid tubercle (Bottom) Areas of erosion in the hyaline cartilage in the head of the glenoid cavity, exposing the subchondral bone

period and indicates loss of elasticity. With increasing age this alteration in the articular cartilage became more apparent and increased in intensity, reaching its height of progression in the sixth decade. In all instances the lesion was more marked in

the region of the comma's head. Simultaneously, small distinct furrows, fibrillation of the superficial layers of the cartilage and softening appeared in and immediately below the central circular area of the head of the comma, especially along its ventral



FIG. 53 A circular area is barely visible in the center of the head of the comma. This is a specimen of the second decade. In the subscapularis and the coracohumeral areas of the synovial membrane are fingerlike synovial villi projecting into the lumen of the joint cavity



FIG. 55 Degenerative changes in the hyaline cartilage in the center of the head of the comma, with formation of connective tissue which assumes the characteristics of imperfectly formed fibrocartilage. This is a specimen of the fifth decade. ($\times 136$)

ity in each successive decade leading one to believe that this circular area might be one of greater contact than the surrounding articular cartilage. According to Fick, Poirier was the first to make this observation. However he believed this region of the comma's head to be covered with fibrocartilage. The findings in this study do not substantiate this observation. Microscopic section through the area in question of infant shoulders revealed that hyaline cartilage was the articular tissue found in this region (Fig. 54). In specimens of later decades the severe degenerative alterations of the cartilage in the center of the comma's head and metaplasia of the degenerated hyaline cartilage to imperfectly formed fibrocartilage explain how such an erroneous interpretation may be made (Fig. 55).

Macroscopically alterations were noted as stated in the articular cartilage of the glenoid cavity as early as the second decade. The bluish white glistening resilient delicate articular cartilage found in infancy gave way to a firm compact, opaque tissue demonstrable as early as the second age



FIG. 54 Typical hyaline cartilage found in the center of the head of the comma in shoulders of infants. (Photomicrograph $\times 67$)

tive joints in each decade disclosed a gradual, steady progression of pathologic alterations of the articular cartilage, paralleling increasing age. Normally, the superficial stratum of hyaline articular cartilage reveals many tightly packed spindle-shaped cartilage cells in a dense matrix whose fibrils are arranged parallel with the joint cavity. The earliest lesion noted was fibrillation of the superficial layers of the cartilaginous matrix of the hyaline cartilage parallel with the joint space. This change was first noted in the second decade.

Fibrillation in the deeper strata of the cartilage extending for variable distances was observed to be at right angles to the joint lumen (Fig. 57 *top left*). Only the transitional layer of the cartilage down to the calcified zone was involved in specimens of the earlier decades. However, from the fifth decade on, the process extended to the subchondral bone thereby exposing it in many instances.

Cartilage cells adjacent to the fibrillated matrix demonstrated evidence of degeneration. The cells were fewer in number below the tangential stratum. They appeared to be swollen and their nuclei stained abnormally. The normal pattern of the cells in the various layers was lost. The cells in the transitional and calcified zones, although fewer in number, were arranged in groups of varying size.

Microscopic study disclosed the progressive nature of the lesions described above with the maximum alterations appearing in the sixth decade (Fig. 57 *bottom left*). Lesions of the seventh, the eighth and the ninth decades demonstrated no appreciable increase in severity over those of the previous age periods.

In this investigation subchondral bony thickening in the ventral and inferior regions of the head of the comma appeared as a frequent abnormality after the fourth decade. Thickening was also disclosed in the center of the comma's head after the fifth decade. The cartilage overlying the above areas revealed advanced degenerative alter-

ations in most instances. Evidence of subchondral bony thickening was a rare feature in the tail of the comma where the cartilage lesions were seldom severe. This was a constant finding.

Histologic examination of sections through the head of the comma in the earlier decades demonstrated only minimal alterations in the subchondral trabeculae. As a rule, the architecture of the cancellous bone appeared to be normal. Occasionally, light thickening and indentation of the end plate of the subchondral bone was discernible. As a rule, the cartilage over the above areas revealed marked fibrillation, fraying and thinning.

In later decades, immediately adjacent to the deep layers of the degenerative hyaline cartilage in the center of the comma's head and to the thinned zone of calcination of the hyaline cartilage in its ventral and inferior regions, the bony trabeculae were hypertrophied, dense and compact. The marrow spaces were small and contained vascular connective tissue. Endosteal cells or osteoblasts lining the trabeculae were discernible in large numbers exhibiting evidence of multiplication and proliferation of endosteal tissue thereby increasing the thickness of the trabeculae by adding newly formed osteoid tissue upon their surfaces (Fig. 57 *bottom right*). On several occasions small discrete islets of cartilage were observed in the marrow spaces. This study affords no original explanation for their origin or significance.

In this series marginal proliferation of bone and cartilage was discernible grossly in the region of the tail of the comma in two specimens of the third decade. The marginal elevation was more or less symmetrical and involved the entire articular periphery of the comma's tail. It was noted that this abnormality increased in frequency and became more prominent in the subsequent decades. The labrum glenoidale overlying the rim of the glenoid cavity obscured to a great degree the extent of the lesion in the inferior and ventral regions of the head

and inferior margins where an increase in thickness of the cartilage was also noted.

The above lesions have been observed and described adequately by many students of this subject (Bennett and Bauer 1931, Keyes 1933, Parker, Keefer, Myers, and Irwin 1934, Nichols and Richardson 1909, Bennett and Bauer 1936). These alterations in the successive decades revealed a gradual rise in magnitude the maximum changes being observed in the sixth age period. Concurrently the hyaline cartilage of the tail of the comma became involved but to a lesser degree than that of the comma's head. With the rise in severity of these pathologic changes the cartilage assumed a papillary or velvetlike appearance (Fig. 56 *top*). Occasionally in the older

age periods due to thinning, fibrillation of cartilage and loss of compact tissue, small irregular, scalloped ulcerated areas were discernible in the articular cartilage, especially in the central inferior and ventral aspects of the comma's head. These exposed the subchondral bone which was thickened but never attained a highly polished surface such as described in weight bearing surfaces of the knee joint by Bennett and Bauer 1936, Nichols and Richardson 1909, and Ely 1923 (Fig. 56 *bottom*). Although such lesions were numerous and encountered frequently in this study they never were so extensive as to involve large areas of the glenoid cavity whereby large portions of the subchondral bone were exposed.

Microscopic examination of representa-

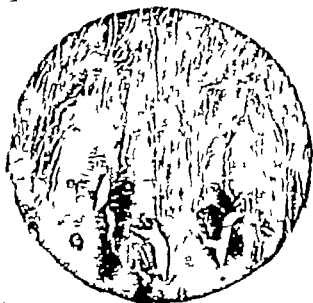
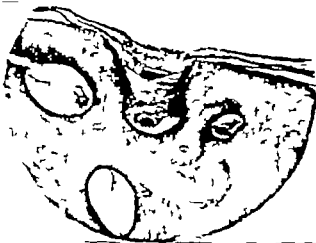


FIG. 57 (*Top*) Fibrillation of the superficial layers and vertical splitting of the deeper layers of the hyaline cartilage in the center of the head of the comma. (*Bottom left*) Marked degenerative changes of the hyaline cartilage in the head of the comma in a specimen of the sixth decade. Note (1) connective tissue proliferation and metaplasia of hyaline cartilage to imperfectly formed fibrocartilage and (2) grouping of cartilage cells in degenerating cartilage ($\times 67$). (*Bottom right*) Complete degeneration of the hyaline cartilage marked thickening of the subchondral bone, narrowing of the marrow spaces and outgrowth of connective tissue from marrow spaces covering the denuded bony surface. (Photomicrograph $\times 67$)



tive joints in each decade disclosed a gradual, steady progression of pathologic alterations of the articular cartilage paralleling increasing age. Normally, the superficial stratum of hyaline articular cartilage reveals many tightly packed spindle-shaped cartilage cells in a dense matrix whose fibrils are arranged parallel with the joint cavity. The earliest lesion noted was fibrillation of the superficial layers of the cartilaginous matrix of the hyaline cartilage parallel with the joint space. This change was first noted in the second decade.

Fibrillation in the deeper strata of the cartilage extending for variable distances, was observed to be at right angles to the joint lumen (Fig 57 *top left*). Only the transitional layer of the cartilage down to the calcified zone was involved in specimens of the earlier decades. However, from the fifth decade on, the process extended to the subchondral bone thereby exposing it in many instances.

Cartilage cells adjacent to the fibrillated matrix demonstrated evidence of degeneration. The cells were fewer in number below the tangential stratum. They appeared to be swollen and their nuclei stained abnormally. The normal pattern of the cells in the various layers was lost. The cells in the transitional and calcified zones although fewer in number were arranged in groups of varying size.

Microscopic study disclosed the progressive nature of the lesions described above with the maximum alterations appearing in the sixth decade (Fig 57 *bottom left*). Lesions of the seventh, the eighth and the ninth decades demonstrated no appreciable increase in severity over those of the previous age periods.

In this investigation subchondral bony thickening in the ventral and inferior regions of the head of the comma appeared as a frequent abnormality after the fourth decade. Thickening was also disclosed in the center of the comma's head after the fifth decade. The cartilage overlying the above areas revealed advanced degenerative alter-

ations in most instances. Evidence of subchondral bony thickening was a rare feature in the tail of the comma where the cartilage lesions were seldom severe. This was a constant finding.

Histologic examination of sections through the head of the comma in the earlier decades demonstrated only minimal alterations in the subchondral trabeculae. As a rule the architecture of the cancellous bone appeared to be normal. Occasionally slight thickening and indentation of the end plate of the subchondral bone was discernible. As a rule the cartilage over the above areas revealed marked fibrillation, fraying and thinning.

In later decades immediately adjacent to the deep layers of the degenerative hyaline cartilage in the center of the comma's head and to the thinned zone of calcification of the hyaline cartilage in its ventral and inferior regions the bony trabeculae were hypertrophied, dense and compact. The marrow spaces were small and contained vascular connective tissue. Endosteal cells or osteoblasts lining the trabeculae were discernible in large numbers exhibiting evidence of multiplication and proliferation of endosteal tissue, thereby increasing the thickness of the trabeculae by adding newly formed osteoid tissue upon their surfaces (Fig 57, *bottom right*). On several occasions small discrete islets of cartilage were observed in the marrow spaces. This study affords no original explanation for their origin or significance.

In this series marginal proliferation of bone and cartilage was discernible grossly in the region of the tail of the comma in two specimens of the third decade. The marginal elevation was more or less symmetrical and involved the entire articular periphery of the comma's tail. It was noted that this abnormality increased in frequency and became more prominent in the subsequent decades. The labrum glenoidale overlying the rim of the glenoid cavity obscured to a great degree the extent of the lesion in the inferior and ventral regions of the head

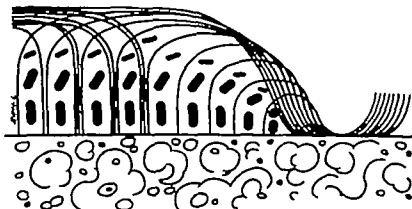


FIG 58 Note advanced marginal lippings along the rim of the glenoid cavities. The new bone formation is more pronounced along the anterior and inferior portions of the glenoid cavities.

of the comma. However investigation of a large series of scapulae denuded of all soft tissue disclosed the maximum degree of new bone formation to be located along the ventral border of the comma especially in its lower region and in the inferior margin of the comma's head (Fig 58). Some degree of macroscopic marginal lipping was noted in all specimens after the third and the fourth decenniums. It was interesting and also of considerable significance to note that there was a close parallelism between the degree of labral detachment and degenerative changes and the prominence of peripheral bone and cartilage formation.

The nature and the severity of the alterations detected in the articular cartilage and subchondral bone in the center and the in-

ferior regions of the head of the comma, permit one to conclude that the above areas sustained the greatest functional strain and were areas of greater contact than the remaining portions of the glenoid cavity. On the other hand, marginal lipping, especially in the older specimens was found to be a very striking and pronounced alteration located in regions which withstand only minimal pressure. It became obvious therefore that a factor other than direct pressure played a greater role in the formation of these peripheral excrescences. The anatomic arrangement of the intracapsular elements of the shoulder joint the relationship of the synovialis and capsule to cartilage and bone at the periphery of the glenoid cavity and the functional demands made



Architestructural pattern of collagenous fibers in cartilage (after Benninghoff)

FIG 59 The structural pattern of the collagenous fibers in cartilage as worked out by Benninghoff. Note that the fibers at the periphery of the cartilage do not end in the deeper layers of cartilage but are continuous with the fibers of the capsule.



FIG 60 Synovial membrane lining the labrum glenoidale. The fibers of the synovialis merge with the superficial fibers of the fibrocartilage but fail to reach the hyaline cartilage of the glenoid cavity. Note how the fibers of capsule on the right are continuous with the fibers of the labrum on the left ($\times 146$)

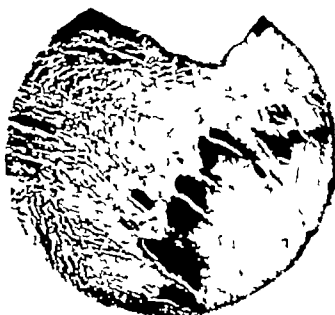


FIG 61 The formation of new bone from the periphery of the glenoid cavity on the right immediately beneath a degenerating labrum glenoidale on the left. Labrum is beginning to tear away from the glenoid rim ($\times 67$)

on this joint may provide a reasonable explanation

Benninghoff (1925) deciphered the structural pattern of hyaline cartilage and correlated it with function. He described a system of radial fibrils which arose from the calcified layer of cartilage, ascended radially through the deep and transitional layers to reach the superficial zone, where they ran in a tangential course for varying distances then were deflected into the deeper layers to end in the calcified zone. At the periphery of the cartilage the outer segments of the radial fibrils were grouped into a dense layer which became continuous with the fibrous capsule and collagenous fibers of the synovialis. Such an arrangement affords a transitional zone of connective tissue between the articular cartilage and the fibrous and synovial capsule and can be considered as a functional adaptation in response to forces of stress and traction at the peripheral joint regions during joint function (Fig 59). However, the relationship of the glenoid cavity and its articular capsule differs from that described above. The fibrocartilaginous labrum is interposed between the hyaline cartilage and the

fibrous capsule. Its glenoid surface is continuous with the fibrous capsule. The synovialis, which lines the fibrous capsule projects for varying distances over the labrum and blends with its superficial fibers, but fails to reach the articular cartilage (Fig 60). Also, the outer fibers of the capsule merge with the periosteum below the inferior edges of the labrum glenoidale. This region, even in the later age periods, is richly supplied with blood vessels.

In this study, microscopic investigation revealed that the most common modes of genesis of marginal proliferations were (1) formation, condensation and projection of subchondral bone into the marginal articular cartilage adjacent to the glenoid surface of the labrum and (2) periosteal bone formation (Fig 61). In some specimens still another mode of bone formation was disclosed. Metaplasia of vascular proliferating connective tissue into islands of new bone and imperfectly formed fibrocartilage and hyaline cartilage were observed in the transitional bone between the outer capsular



FIG 62 (*Left*) Note that the fibers of the cartilage (below) are continuous with those of the labrum glenoidale (above) ($\times 67$)

FIG 63 (*Right*) Early separation of labrum from glenoid brim. Note that line of cleavage is in the fibers of the labrum close to the glenoid brim.

fibers and the periosteum. The prominence of the marginal ridgings paralleled the severity of the alterations in the labrum and the synovialis rather than the changes in the articular cartilage.

These observations are compatible with the concept that marginal outgrowths are in response to functional stresses chiefly traction on the outer joint margins. Traction and shearing forces acting on the periphery of the glenoid cavity are derived from several sources. The fibrous capsule of the glenohumeral joint is a loose redundant structure which allows the humeral head a wide range of motion. Thus it is apparent that at the ends of the arcs of motion traction and shearing forces are applied at the peripheral joint margins of the glenoid cavity. Furthermore the weight of the upper extremity in a dependent position exerts a definite traction force through the articular capsule at its insertion into the labrum glenoidale and glenoid rim. In addition to the above there are two other important factors which are responsible for the formation of marginal excrescences. The biceps

tendon which is continuous with the superior posterior portion of the labrum glenoidale exerts a distraction force at the glenoid rim in the region of the comma's tail during normal joint function. Likewise the glenohumeral ligaments, which in most instances are attached to the superior and the anterior portions of the labrum maintain sustained traction upon the labrum at the end of the arc of external rotation, thereby tending to pull the labrum from its bony and cartilaginous attachments. All these factors furnish stimuli through the media of traction and distraction forces which induce metaplasia of connective tissue in transitional zones, hypertrophy of subchondral bone and periosteal bone formation.

LABRUM GLENOIDALE AND BICEPS TENDON

The labrum glenoidale is a triangular fibrocartilaginous structure attached by its base to the rim of the glenoid cavity. Its glenoid surface is continuous with the hyaline cartilage of the glenoid cavity while its capsular surface blends with the capsule in

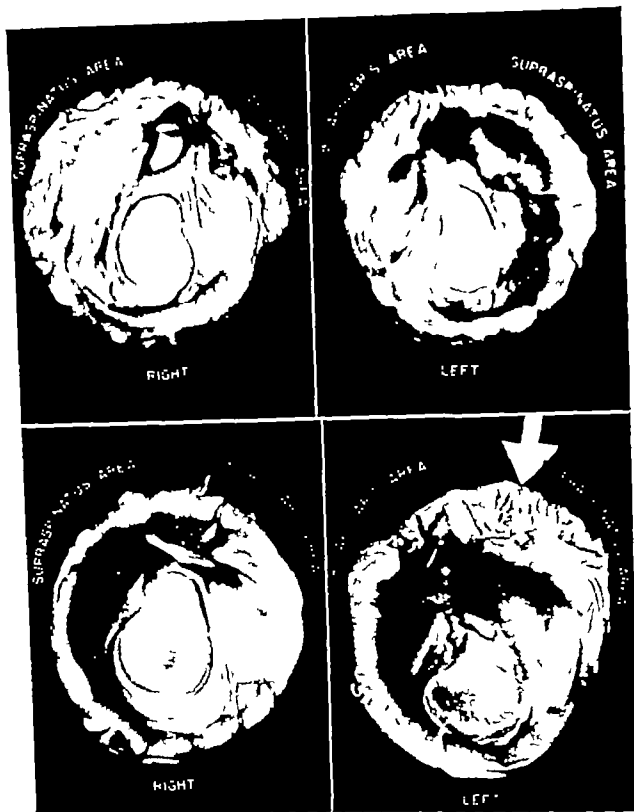


FIG 64 (*Top left*) Early detachment of the labrum from the tail of the comma in a specimen of the second decade. (*Top right*) Further detachment of the labrum from the tail of the comma in a specimen of the third decade. (*Bottom left*) Detachment of the labrum from the glenoid cavity in the region of the tail of the comma in a specimen of the fourth decade. Note the well-defined, circular area in the center of the head of the comma. (*Bottom right*) Complete detachment of the labrum from the tail of the comma and the anterior aspect of the glenoid rim in a specimen of the sixth decade. Also note marked thickening of the labrum in the region of the tail of the comma, and thinning of the labrum in the region of the head of the comma where numerous tabs and fringes of synovial and connective tissue are also evident. Arrow points to a huge incomplete tear in the supraspinatus and the infraspinatus tendons.

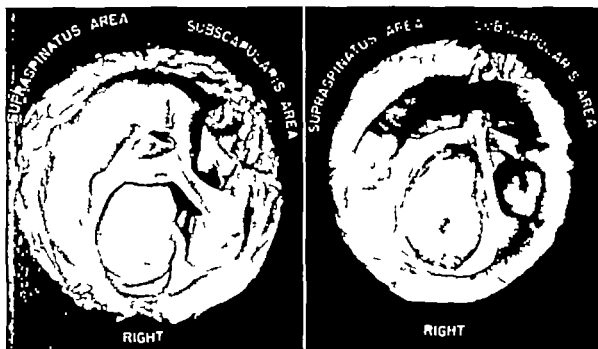


FIG 65 (Left) Pronounced detachment of labrum in a specimen of the seventh decade. (Right) Labrum detachment in specimen of seventh decade.

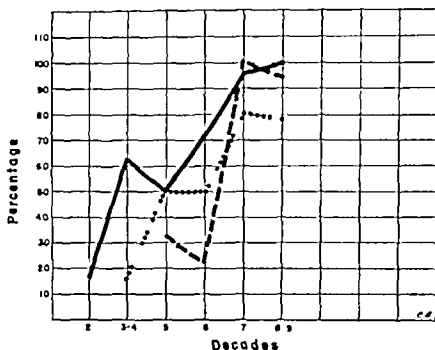
the region of the comma's head. However that portion around the tail of the comma either may be continuous with the capsule or entirely free (Fig 50). Grossly the structure in most specimens of the second decade was smooth and glistening; its edges were sharply defined and it adhered closely to the rim of the glenoid cavity.

Microscopic examination of shoulders obtained from infants and from 6-7 and 8 month fetuses revealed the collagenous fibers of the labrum to be continuous with those of the hyaline cartilage on its glenoid surface and with those of the fibrous capsule on its capsular surface (Fig 62). The synovialis covers the outer portion of its glenoid surface but fails to extend as far as the hyaline cartilage (Fig 60).

Alterations in the labrum were observed as early as the second decade. They consisted of slight wrinkling and unevenness of the capsular surface and gross evidence of detachment of its glenoid border from the hyaline cartilage of the glenoid cavity in the region of the tail of the comma (Fig 64). Microscopic examination of specimens of the second decade exhibit a gradual tearing

away of the labrum from the glenoid rim. The line of cleavage appears to be through the fibrocartilaginous fibers of the labrum close to the glenoid rim (Fig 63). Such alterations were encountered more frequently and likewise were more pronounced with increasing age. In the second decade 16.6 per cent of the specimens demonstrated some degree of nonattachment in the region of the comma's tail; in the third and the fourth decades 63.6 per cent; in the fifth decade 50 per cent; in the sixth decade 72.2 per cent; in the seventh decade 96.4 per cent; and in the eighth and the ninth decades 100 per cent (Fig 66). From the above observations it is reasonable to conclude that detachment of the labrum is associated with advancing age. Moreover the labrum's intimate relationship to the glenohumeral ligaments and the biceps tendon renders it vulnerable to stresses (chiefly to distracting forces) which play a major role in the production of areas of nonattachment.

In the light of this information, it is difficult to conceive that labral and capsular defects in the anterior portion of the shoul-



- Labral detachments in each decade
 Biceps tendons in each decade showing degenerative changes
 ---- Tears in fibrotendinous cuff in each decade

FIG 66 Rise in severity of degenerative changes in the biceps tendon and in the musculotendinous cuff and the increase in the number of labral detachments in successive decades



FIG 67 (Left) Marked hyperplasia of detached labrum in the region of the tail of the comma and advanced hypertrophy of all the glenohumeral ligaments. There are numerous fringes in the tail region between the hypertrophied labrum and the glenoid cavity

FIG 68 (Right) Synovial villi, connective tissue tabs and fringes arising from the labrum at the point of detachment from the glenoid cavity ($\times 71$)

der joint are the responsible factors for recurrent dislocations of the shoulder joint. Clinical experience notes that recurrent dislocations are common in the earlier decades but rare after the fourth decade, yet this study reveals that the greatest number and the most severe detachments occur in those age periods in which this lesion is only rarely encountered. Therefore it is obvious that the agents which play an even greater role in the production of recurrent dislocations of the shoulder joint are other than labral and capsular lesions as described by Bankart, Nicola and many others.

On the other hand, one can understand readily why the percentage of failures (36 to 40 per cent) is so high when the Nicola procedure or its numerous modifications are used to stabilize recurrent dislocating shoulders. The biceps tendon is attached firmly to the apex of the labrum glenoidale. By virtue of a distracting force upon the labrum incident to joint function it aids in pulling the labrum from its attachment on the glenoid rim. Should the arm be suspended from the apex of the glenoid cavity as is done in the various intracapsular and extra capsular procedures (by anchoring the biceps tendon to the humeral head) the added pull upon the labrum enhances its detachment from the glenoid cavity thereby defeating the very purpose for which the operation was done originally to stabilize the humeral head by the creation of an intracapsular ligament.

Hypertrophy of the labrum in the region of the comma's tail was demonstrable as early as the second age period. This alteration increased in frequency and degree with each subsequent decennium. In fact only one specimen of the third and the fourth decades did not reveal evidence of hyperplasia. Tabs and fringes especially along the ventral portion of the glenoid border of the labrum first observed in the second decade were frequent in later age groups and were associated with hypertrophy of the labrum. They were more pronounced in

those specimens showing large areas of non attachment. There seems to be a close parallelism between tabs, fringes and villous formation and the degree of labral detachment. Specimens of the seventh, the eighth and the ninth decades exhibited the above mentioned alterations in their severest forms (Fig. 67).

Microscopic study discloses that tabs, fringes and villi arise from several sources. They may originate from remnants of fibrocartilage on the glenoid border of the labrum or from those remnants still attached to the hyaline cartilage of the glenoid cavity, from the transitional zone tissue between the fibrous capsule and the perichondrium of the glenoid cavity from the bone marrow spaces of the glenoid and from the synovialis covering the outer border of the glenoid surface of the labrum glenoidale (Fig. 68).

In the region of the comma's head labral degenerative changes associated with senescence differ from those in the tail region. Whereas hypertrophy of the fibrocartilaginous structure was the outstanding change in the labrum in the tail of the comma, thinning, fraying and shredding were the usual alterations encountered in the head portion (Fig. 64, bottom right). These changes were first manifested in some specimens of the fifth age period. The alterations were frequent, progressing steadily in severity in specimens of the subsequent age groups. However such abnormalities were noted also in the anterior portion of the labrum of the tail of the comma in several shoulder joints of the fifth decade and in a few of the following decades. Tabs, fringes and villi were also common alterations noted in this region and not a few specimens demonstrated deposition of calcareous material in the fibers of the labrum and degenerated cartilage in the region of the head of the comma. It becomes apparent that such attritional changes are associated with increasing age; also they are the result of constant friction between the comma's head

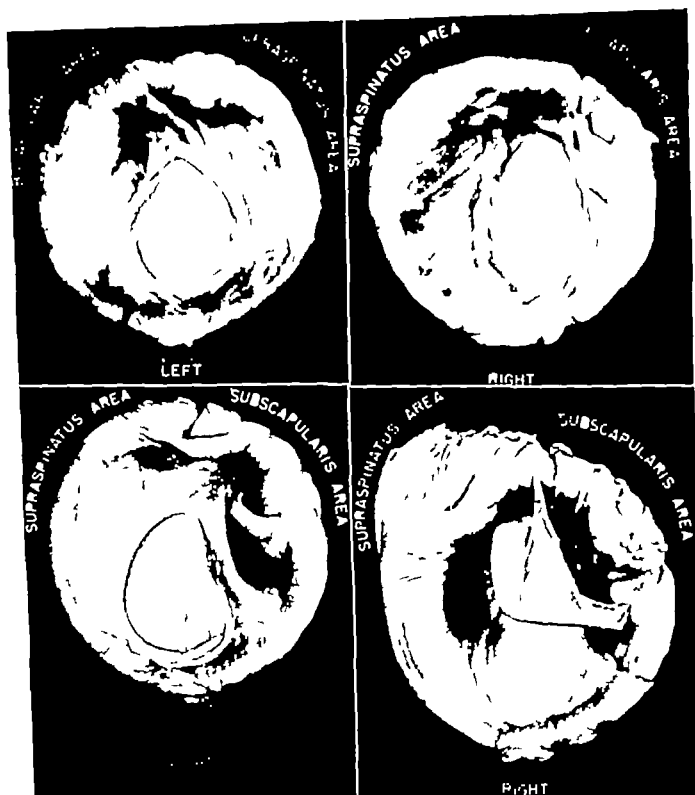


FIG 69 (Top left) A double biceps tendon (Top right) Absence of a biceps tendon and a firmly attached labrum to the glenoid rim (Bottom left) The biceps tendon is extrasynovial lying within the fibrous capsule. This structure has failed to migrate to an intracapsular position. Note the firm attachment of the labrum to the rim of the glenoid cavity. The inferior glenohumeral ligament in this specimen is well defined and extends from the subscapular area to the triceps area. (Bottom right) A well formed mesentery of the biceps tendon and a large incomplete tear in the musculotendinous cuff in the supraspinatus area, proximal to which there is a distinct hypertrophied falciform ligament.

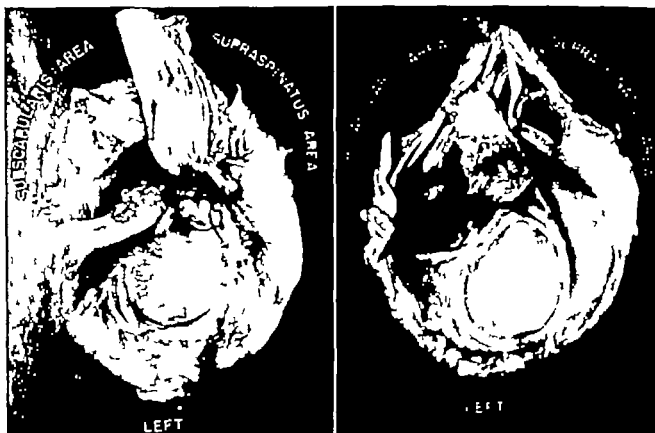


FIG 70 (Left) Marked hypertrophy of all the intracapsular structures except the labrum in the region of the comma's head also advanced fraying shredding and hypertrophy of the biceps tendon Note the advanced degenerative lesions in the musculotendinous cuff of this specimen.

FIG 71 (Right) A specimen with complete avulsion of the musculotendinous cuff, advanced degenerative changes and hypertrophy of the biceps tendon

and articulating surface of the humerus for evidence points to the fact that this area may be one of greater contact than elsewhere

The intracapsular portion of the biceps tendon occupies a relatively constant position in the superior and slightly anterior region of the shoulder joint It sends fibers into the superior posterior portion of the labrum and in many instances it is attached to the superior glenohumeral ligament occasionally to the middle ligament and (in a few instances) to all three glenohumeral ligaments

Several specimens exhibited various developmental abnormalities of the tendon In Figure 56 *top left* the tendon occupied a partially intracapsular position In Figure 69 *top left* it existed as a double structure In Figure 69 *top right* it was absent and

in Figure 69 *bottom left* it was entirely within the fibrous capsule Several tendons possessed mesenteries (Fig 69, *bottom right*) No gross degenerative abnormalities were manifest in the biceps tendons until the fourth decade One tendon in this group revealed evidence of widening and thickening However the frequency and the prominence of these alterations showed a progressive rise in the subsequent decades In the fifth decennium 50 per cent of the tendons revealed the above changes in the sixth 50 per cent in the seventh 80 per cent and in the eighth and the ninth, 78 per cent It was interesting and of some significance to note that the tendons which showed the greatest pathologic disorders were found in specimens which demonstrated severe alterations in the fibrotendinous cuff (Figs 70 and 71) This observation leads one to be-



FIG 72 Synovial tabs and fringes in the coracohumeral area and a middle glenohumeral ligament which is not continuous with the labrum.

lieve that with severe degenerative changes in the rotator cuff, the biceps tendon may be forced to assume the role of the main support for the upper extremity and may be subjected to great attritional forces, thereby accounting in part for the above changes.

Many tendons of the later decades revealed marked fraying, shredding and disorganization, in one shoulder joint it was absent. Only a small stub of the intracapsular portion remained (Fig 56 *top right*). The intertubercular groove of the corresponding humerus of this specimen was completely obliterated, while the proximal end of the extracapsular portion of the tendon had attained a bony anchorage on the humeral shaft below the level of the intertubercular sulcus.

SYNOVIAL MEMBRANE

Grossly the synovialis of specimens of the early decades demonstrated several distinct features. In the bursal recesses and



FIG 73 A typical synovial villus and hypertrophy of the synovial membrane ($\times 145$)

coracohumeral areas it existed as a more-or-less redundant structure from whose surface small fringes, tabs or villi project into the joint cavity (Fig 72). Its inner layer consists of densely packed oval or stellate connective tissue cells, elastic fibers, numerous fat cells, scanty matrix and a rich blood supply. It merges with an outer fibrous layer rich in collagenous substance but poor in cellular elements. The synovial membrane covering the intracapsular elements (glenohumeral ligaments, labrum glenoidale and biceps tendon) is thin, smooth, glistening and closely adherent to the underlying structures (Fig 50). Its inner surface is very thin and delicate, in some areas comprising a single layer of loosely packed flattened cells with little or no subintimal connective tissue.

Villous formation and proliferation are indisputable alterations associated with advancing age, although one must concede that other factors such as mechanical and chemical irritation of the synovialis, also may be responsible for these changes. They may exist as fine fingerlike projections or as branching papillary or even polypoid structures. Their histologic structure con-

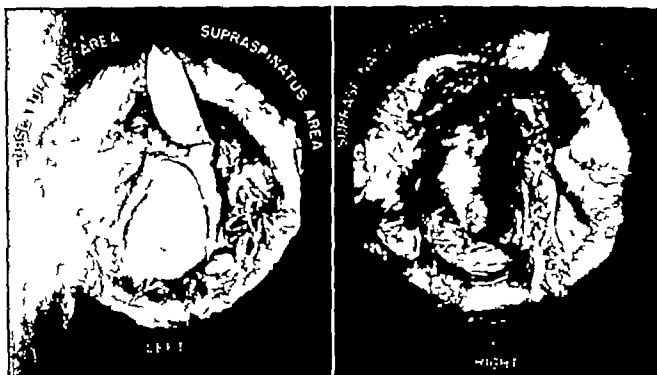


FIG 74 Advanced villous hypertrophy and proliferation of the synovial membrane. Also thickening of the intracapsular structures so that the synovial recesses are practically obliterated. The corresponding humeral heads of both these specimens exhibited evidence of severe trauma.

sists essentially of a fine connective tissue core containing fat cells and capillary blood vessels and enclosed by enveloping layers of specialized connective tissue (synovial cells). Lymphoid and plasma cells are present in varying numbers within the connective tissue stalks (Fig 73). In this investigation villi were detected as early as the second decade in the subscapularis and coracohumeral areas (Figs 53 and 72).

These abnormalities of the synovial membrane were observed frequently in many specimens of the third, the fourth and the fifth decades. Although relatively slight there was a definite increase in gradient up to the sixth decennium. Moreover in many specimens of the fourth and the fifth age periods the above alterations were not only disclosed in the subscapularis and coracohumeral regions of the joint but also in the other areas of the synovial membranes especially in the infraspinatus and teres minor and triceps areas. Generally speaking

villous projections and proliferations in specimens of the sixth, the seventh, the eighth and the ninth decades surpassed in prominence those noted in the previous age periods.

Figure 74 demonstrates the effect of trauma on the synovials. The corresponding humeral heads of these shoulder joints disclosed marked irregularity of their articular surfaces secondary to malunited fractures of the anatomic necks. The resulting incongruity of the articulating surfaces inflicted repeated trauma to the synovial tissues. The trauma was undoubtedly responsible for the reactionary response noted in the synovial membranes of the above shoulder joints. As the photographs show both the shoulder joints revealed numerous villi of varying sizes and shapes arising from all fields of the synovial membrane; however the villi were more pronounced in the infraspinatus and teres minor area. Many of the villi were rounded, thickened,



FIG 75 (*Left*) Numerous tabs and fringes between the labrum and the glenoid cavity, as if an attempt were being made to reattach the labrum to the glenoid rim.



FIG 76 (*Right*) Calcareous deposits in the degenerated cartilage in the head of the comma and tabs and fringes covering surface of the labrum. In the supraspinatus area, a large defect exists in the musculotendinous cuff, proximal to which there is a well formed falciform ligament.

stubby and of yellow color being heavily laden with fat cells while others were fine, fingerlike branching projections

Although both joints presented advanced synovial impairment the articular cartilage in Figure 74 *left* exhibited only minimal alterations while that in Figure 74 *right* far advanced degenerative changes. This last observation supports the concept that degenerative articular changes are primary and not secondary to synovial abnormalities. As a rule however specimens which showed great impairment of the articular cartilage also demonstrated severe lesions of the synovial lining. Secondary changes were not infrequently noted in villi especially in the later decades. The most common alteration was hyaline degeneration of the connective tissue in some instances, formation of imperfect cartilage was observed

Tabs and fringes, arising from the labrum glenoidale and the glenoid rim were observed in some specimens of the second decade. They increased in size and number from decade to decade. Only two specimens in the fifth decade failed to show these alterations; they were present in all specimens of the sixth, the seventh, the eighth and the ninth age periods. As previously stated, such abnormalities were associated chiefly with detached labra. Grossly and microscopically, they originated from the synovial lining over the labrum, from the glenoid border of the labrum and from the periphery of the glenoid rim. As a rule they were concentrated in the region of the tail of the comma and along the ventral margin of the glenoid cavity in which areas labral detachments were most commonly observed. There was a close parallelism between the prominence of these changes and the degree



FIG 77 Advanced degeneration of the synovial membrane in the falciform ligament. Note marked fibrous tissue proliferation and hyalinization of this tissue ($\times 67$)

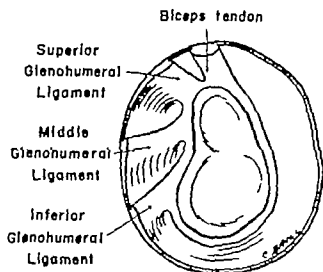


FIG 78 Schematic representation of superior middle and inferior glenohumeral ligaments. They converge from the humerus toward the ventral border of the glenoid cavity, to be continuous with the labrum and the biceps tendon

of labral detachment. Microscopically, the tabs arising from the capsular border of the labrum consisted of irregularly shaped synovial villi of varying sizes similar to those arising from the synovial membrane in the subscapularis region. Those originating from the glenoid rim and fibrocartilage consisted of irregular frayed portions of fibrocartilage many of which were undergoing hyalinization. Moreover, some of these tabs and fringes were composed of connective tissue arising from the vascular transitional zone between the fibrous capsule and the perichondrium while others arose directly from the peripheral marrow spaces of the cancellous bone. In the later decades many specimens revealed the space between the glenoid rim and detached labrum to be obliterated partially or completely by numerous tabs and fringes as if an attempt were being made to reattach the labrum to the glenoid rim (Figs 75 and 76). These degenerative structures showed many secondary changes especially in the older specimens. The changes comprised chiefly hyalinization and calcareous material within these projections (Fig 76).

Hypertrophy of the synovial membrane, due to fibrosis of the subintimal connective tissue, was noted as early as the third and the fourth decades. In the subsequent age groups there was a gradual, progressive rise in the intensity of this process. The structures which were subjected to constant functional stress disclosed the greatest amount of thickening. Thus there were marked hyperplastic changes in the glenohumeral ligaments, the biceps tendon, the superior portion of the labrum which is continuous with the biceps tendon and the superior glenohumeral ligament and the ventral segment of the labrum which is attached to some or all of the glenohumeral ligaments. In some instances the increase in thickness of the above structures and of the total synovial lining was so great that the synovial pouches in the anterior aspect of the shoulder joint were reduced in size or were obliterated completely (Fig 74).

Macroscopic tears in the synovial membrane were first noted in the fifth decade and they were associated with degenerative defects in the corresponding areas of fibrotendinous cuff. Proximal to the cuff lesion



FIG 79 A well formed middle glenohumeral ligament below the level of the superior glenohumeral ligament. Also a large superior subscapularis recess and a still larger inferior subscapularis recess which may be interpreted erroneously as a rent in the capsule.

the synovial membrane always was found torn (Fig 64, *bottom left*). It became apparent that the synovial and fibrotendinous alterations occurred simultaneously. The proximal edge of the synovial defect became thickened, smooth and glistening and formed a falciform ligament across the defect in the fibrotendinous cuff. In this study the synovial lesions paralleled in number and severity the alterations in the fibrotendinous cuffs. Microscopic study of sections through the above falciform ligaments revealed marked hyperplasia of the subintimal connective tissue and a scarcity of cellular elements (Fig 77). Hyalinization of the synovial membrane was a frequent finding. Not infrequently, metaplasia of the connective tissue of the subintimal stratum resulted in the formation of fibrocartilage.

GLENOHUMERAL LIGAMENTS AND SYNOVIAL RECESSES

The fibrous capsule is reinforced anteriorly by three ligaments designated as the superior, the middle and the inferior gleno-



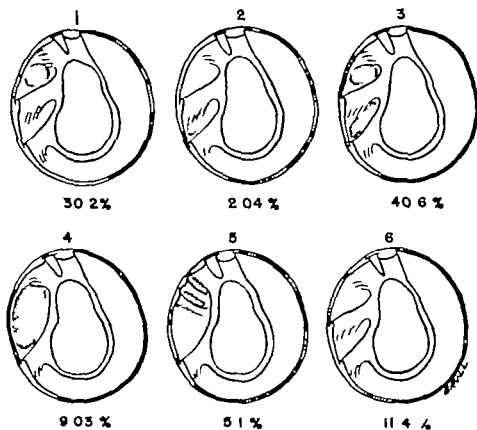
FIG 80 Marked hypertrophy of the synovial and all intracapsular elements tending to obliterate the synovial recesses and the pattern of arrangement of the glenohumeral ligaments.

humeral ligaments (Fig 78) Generally speaking, they converge from the humerus toward the ventral border of the glenoid cavity, to be continuous with the labrum glenoidale and the biceps tendon They limit external rotation by acting as check reins. In the anterior portion of the shoulder joint are found synovial recesses, whose number size and location show considerable variation This variability is dependent largely on the topographic variations of the glenohumeral ligaments

MIDDLE GLENOHUMERAL LIGAMENT

The middle glenohumeral ligament is a variable structure In the 96 specimens of

this study, it was found as a well formed distinct structure in 68, it was poorly defined in 16 and absent in 12 When present it arises in most specimens from the labrum, immediately below the superior ligament, and from the neck of the scapula (Fig 79) It inserts into the lesser tuberosity in relationship with the subscapularis tendon Occasionally, it arises only from the labrum At other times it may have no attachment to it (Fig 72) In a few specimens it exists as a double structure, in the anterior portion of the capsule, having no connection with the labrum, the superior ligament or the scapula Length width and thickness vary considerably Such variability was



Types of arrangements of synovial recesses

FIG 81 Six types of arrangement of the synovial recesses (1) Characterized by one synovial recess above the middle glenohumeral ligament. (2) One synovial recess below the glenohumeral ligament. (3) Two synovial recesses. A superior subscapular recess above the glenohumeral ligament, and an inferior subscapular recess below the glenohumeral ligament. (4) One large synovial recess above the inferior glenohumeral ligament the middle glenohumeral ligament is absent. (5) The middle ligament exists as two small synovial folds. (6) Complete absence of synovial recesses.

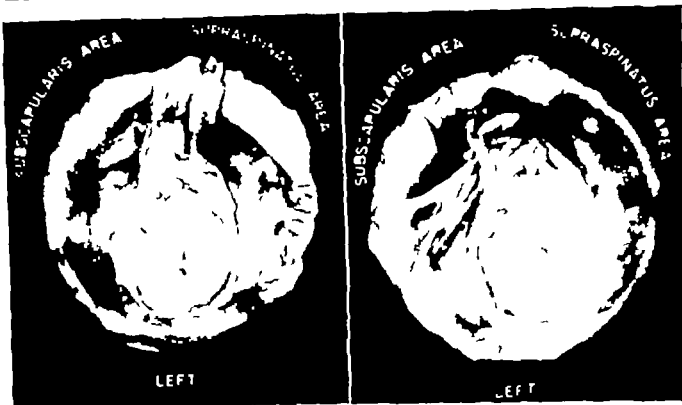


FIG 82 (*Left*) Small synovial recesses above and below the middle glenohumeral ligament. (*Right*) Large synovial recess above the middle glenohumeral ligament. This recess may be interpreted erroneously as a rent in the capsule.

more pronounced in specimens of the later decades. This was due partially to developmental reasons and partially to alterations in the synovial membranes. The marked fibrosis noted in the synovial tissues lining the fibrous capsules was disclosed also in the synovial membranes covering the glenohumeral ligaments. Moreover there was profound hyperplasia of the fibrous tissue elements of the ligaments (Fig 80).

The variable topographic relationship of the glenohumeral ligaments to the synovial recesses gave rise to six distinct variations designated as Types I II III IV V and VI. Type I was observed in 30.2 per cent of the specimens. Type II in 2.04 per cent. Type III in 40.6 per cent. Type IV in 9.03 per cent. Type V in 5.1 per cent. and Type VI in 11.4 per cent (Fig 81). These are developmental types for they were present also in infant shoulders. However because of progressive soft tissue changes the different types may lose their distinguishing features.

Figure 80 revealed such profound alterations in the synovial tissues and all in tracapsular structures that the synovial recesses were indiscernible, and the glenohumeral ligaments were barely distinguishable. One can assume, therefore, that the hyperplastic processes in the synovial tissues and fibrous capsules of these shoulder joints obliterated the recesses, if present, and distorted the distinctive characteristics of the glenohumeral ligaments.

The size of both the superior and the inferior subscapularis recesses, demonstrated extreme variability regardless of the type in which they are found. They may be very small as in Figure 82 *left* or very large as in Figures 82 *right* and 80, *right*. With advancing age there is a tendency for the recesses to become smaller and in some instances, obliterated by increased thickness of the capsular tissues.

Besides physiologic aging, another factor plays a major role in the production of the above findings. It is generally agreed that

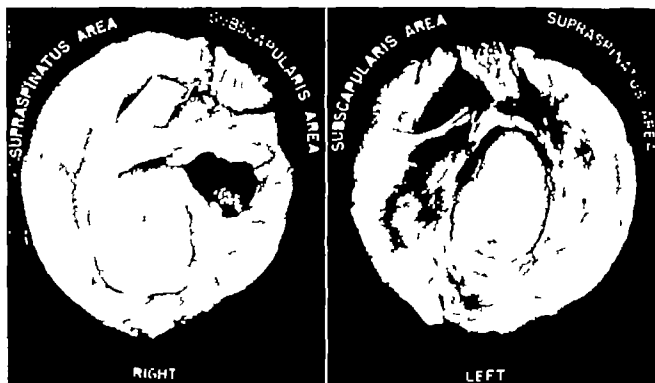


FIG 83 (*Left*) Hypertrophied middle glenohumeral ligament, below which is a large inferior subscapularis recess. The recess may be mistaken for a rent in the capsule, and the middle ligament as a detached labrum.

FIG 84 (*Right*) Ill-defined inferior glenohumeral ligament. It exists as a diffuse thickening of the synovial membrane between the subscapular area and the triceps area.

soft tissues subjected to constant functional stress respond by becoming hypertrophied. The glenohumeral ligaments restrict external rotation of the arm. Therefore it is plausible to assume that over a period of years this function causes hypertrophy of the glenohumeral ligaments.

The anatomic relationship of the middle glenohumeral ligament to the inferior subscapularis recess is of singular clinical significance for it may be interpreted erroneously as a traumatic or pathologic disorder associated with or responsible for recurrent dislocations of a shoulder joint. In the presence of a large recess and a distinct middle ligament, a surgeon exploring the shoulder joint may readily misinterpret the recess as a rent in the capsule and the ligament as a torn and displaced labrum. Figures 79 and 83 clearly illustrate the grounds for such a possibility.

SUPERIOR GLENOHUMERAL LIGAMENT

This structure was the most constant of all the glenohumeral ligaments (Fig 50). It was identified as a distinct structure in 94 specimens and was absent in 2. It arose from the upper pole of the glenoid cavity and the root of the coracoid process. In 73 of the 96 shoulders studied it was found attached to the middle glenohumeral ligament to the biceps tendon and to the labrum in 20 to the biceps tendon and the labrum in 1 to the biceps tendon only and in 2 to the middle ligament the inferior ligament and the labrum, but not to the biceps tendon.

Many years ago Schlemm considered it as the deep portion of the coracohumeral ligament. It inserts in the fovea capitis of the humerus adjacent to the lesser tuberosity in relationship to the portion of the coracohumeral ligament that inserts into

this tuberosity. Frequently, the capsular portion of the superior glenohumeral ligament projected into the lumen of the joint. This condition was constant in the newborn child, in the frog and the guinea pig. Welcher (1877) compared this ligament with the round ligament of the hip joint and found that, during its development, it shifted from an extracapsular to an intra-capsular position. Accordingly, the visibility of this ligament from the synovial side of the capsule will vary.

Like the middle ligament the superior glenohumeral ligament varied considerably in size, although its anatomic position was constant. There was progressive thickening of this structure with increasing age. This change was the direct result of hypertrophy of the synovial and the fibrous capsule associated with senescence. The hyperplastic process may be so advanced that the identity of the structure is obscured.

Ninety-four of the specimens disclosed the superior ligament attached to the labrum. This observation was of some significance for the labrum in the tail of the comma showed evidence of detachment as early as the second decade. The frequency and the degree of this abnormality increased with each subsequent decade. It is reasonable to conclude, therefore, that the superior glenohumeral ligament together with the biceps tendon exerted a distracting force upon the labrum in this region, thus causing this structure to be pulled away from the glenoid cavity.

INFERIOR GLENOHUMERAL LIGAMENT

The inferior glenohumeral ligament is a triangular-shaped structure with its apex at the labrum and its base blending with the capsule between the subscapularis area and the triceps area, thus reinforcing this region (Fig. 69 bottom left). Often it is a very indistinct structure and exists only as a diffuse thickening of the capsule (Fig. 84). In this study it was observed as a well defined ligament in 54 shoulder joints

poorly defined or indistinct in 18 and absent in 24.

Degenerative alterations affecting this structure were similar to those described for the middle and the superior ligaments. They chiefly comprised thickening of the synovial and fibrous elements of the structure. Together with the other two ligamentous structures, it plays an important role in detaching the labrum from its anterior attachment to the glenoid cavity and in the formation of bony excrescences.

MUSCULOTENDINOUS CUFF

The fibrous capsule of the glenohumeral joint is a loose, redundant structure, whose surface area is approximately twice that of the anatomic head of the humerus. Such laxity of the capsule permits wide arcs of motion of the humeral head in all directions. It arises from the margin of the glenoid cavity and the capsular surface of the labrum. In specimens possessing large synovial recesses, the fibrous capsule is not continuous with the labrum glenoidale, anteriorly, but it extends mesially to the base of the coracoid process and then reflects as a very thin structure (consisting of a thin layer of fibrous tissue covered with synovial membrane) along the ventral surface of the neck of the scapula to the labrum (Fig. 50). In the region of the tail of the comma, the capsule usually does not encroach upon the labrum. The tendons of the short rotator muscles (supraspinatus, infraspinatus, teres minor and subscapularis) reinforce the upper half of the fibrous capsule by merging with its fibers. Commingling of the tendon and the capsular fibers is so complete that it is impossible to separate them even by sharp dissection. Close to their insertion into the sulcus of the humerus it is impossible, even microscopically, to distinguish tendon fibers from capsular fibers. Moreover the fibers of the rotator tendons not only interlace with the capsular fibers but also with one another.



FIG. 85 Severe incomplete tears of the musculotendinous cuff in the region of the subscapular area and the supraspinatus and infraspinatus areas.

Gross lesions on the synovial side of the fibrotendinous cuff were first noted in the fifth decade. The alterations consisted of shredding, fraying and tearing of the innermost fibers of the cuff in the regions of the supraspinatus and the subscapularis tendons (Fig. 85). Many specimens of the later decades revealed involvement of the entire thickness of the musculotendinous cuff, thus showing a defect which is referred to clinically as a complete tear.

As recorded previously, the synovial tissues proximal to the defects were always torn, thickened and smooth, forming false ligaments across the defect (Fig. 75). The frequency and the magnitude of these degenerative changes rose steadily with advancing age. In the fifth decade there were cuff tears of some degree in 4 out of 12 shoulder joints; in the sixth in 4 out of 18; in the seventh in 30 out of 30; in the eighth

and the ninth, in 23 out of 24 (Fig. 66). Hitherto, one was prone to believe that the supraspinatus area was most often affected. However, this study disclosed that degenerative alterations are encountered more frequently, though less severely, in the subscapularis region of the musculotendinous structure. Forty-one per cent of the shoulder joints demonstrated lesions in the subscapularis tendon, while 24 per cent showed lesions in the supraspinatus tendon.

Involvement of the infraspinatus tendon was noted in 13 per cent. It was never found as a single lesion but always was associated with a tear of the supraspinatus area. On the other hand, tears in the supraspinatus tendon without extension into the infraspinatus area were recorded in 45.8 per cent of the specimens with lesions in the supraspinatus region of the cuff. Double tears (lesions in both the supraspinatus and the subscapularis regions) were revealed in 34.4 per cent of the specimens, showing gross degenerative changes in the musculotendinous cuff. Two double tears were found in the fifth decade, 2 in the sixth, 7 in the seventh and 10 in the eighth and the ninth. One specimen (Fig. 71) demonstrated an avulsion of the entire musculotendinous cuff. The increase in percentage of tears together with the rise in the magnitude of the lesions in successive age periods indicates that the severe total capsular alterations observed in the seventh, the eighth and the ninth age periods are intimately related with senescence. Although other factors may participate in the production of the above lesions, physiologic aging plays a great role.

Microscopic alterations in the musculotendinous cuff were observed in shoulder joints as early as the third decade. The lesions comprised tearing, fraying and hyalinization of the collagenous fibers, increased vascularity and fibrosis. Specimens of subsequent age periods revealed a gradual advance in gradient of the pathologic changes. The most pronounced lesions were found in the seventh, the eighth and the ninth dec-

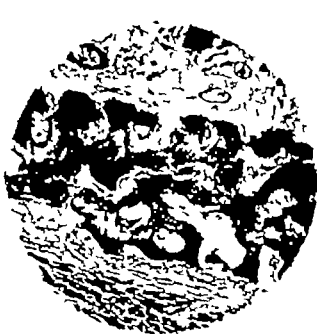


FIG 86 (*Left*) Bone formation within the hypertrophied synovial membrane overlying the musculotendinous cuff in the region of the supraspinatus area. ($\times 65$)

FIG 87 (*Right*) Severe degenerative changes, characterized by shredding and tearing of the fibers of the supraspinatus tendon. Further note transformation of these fibers to fibrocartilage in the region of their point of insertion into the humeral head. ($\times 67$)

ades. After the third age period all shoulder joints disclosed some microscopic impairment in the musculotendinous cuff. As a rule the lesions were encountered more frequently and were of greater severity in the supraspinatus and the subscapularis areas than in the other capsular regions. Metaplasia of connective tissue was not an uncommon observation after the fifth age period. Imperfectly formed fibrocartilage, osteoid tissue and in several specimens bone formation were frequently encountered (Fig 86). The most constant alterations noted in the late age periods were hyalinization and transformation of the fibers of the musculotendinous cuff into fibrocartilage at its point of attachment to the humeral head (Fig 87).

The above observations referable to the fibrous capsule of the shoulder joint differ from those recorded on the knee joint by Nichols and Richardson who observed that the capsule is not thickened, although the fibrous tissue of which it is composed may be exceedingly dense. Parker, Keefer, Myers and Irwin noted that "the capsule of

the joint was as a rule, not thickened although in the cases in which the capsule was stretched the fibrous tissue was extremely tough and of increased density.

In this study, the degree of impairment found in the capsular elements generally surpassed those in the bony and cartilaginous elements of the shoulder joints. Moreover, whereas the maximum level of pathologic changes in the cartilage was attained in the sixth decade, the capsular lesions showed a steady increase in gradient to the ninth decennium.

Although aging is a major factor, it becomes apparent that other agents participate in the production of advanced changes observed in many specimens of the seventh, the eighth and the ninth decades. The musculotendinous portion of the capsule of the shoulder joint possesses a unique anatomic position. It occupies a site between the acromion and the humeral head. Thus it is vulnerable to repeated injuries by being compressed between these two bony points during various positions of abduction and elevation of the arm. Stevens further

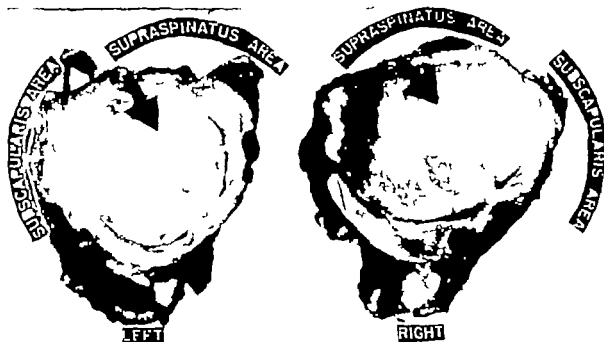


FIG 88 Paired specimens from an individual 48 years of age. Arrows point to the irregularities in the floor of the subacromial bursa. This is a view looking directly on top of the bursa the roof of the bursa has been excised in both specimens. Degenerative areas are observed at points making greater contact with the acromion

noted that failure of the rotator muscles to stabilize the humeral head against the glenoid cavity during abduction causes an upward thrust of the head against the inferior surface of the acromion thereby pinching and compressing the intervening soft tissue structures. Moreover the rotator apparatus functions as the short arm of a long lever. This predisposes the musculotendinous cuff to repeated strain. Strains are produced also by various occupations which demand that the arm function in slightly abducted positions. The accumulative effect of slight repeated traumata over many years is partially responsible for the advanced degenerative and attritional changes in capsular tissues of the shoulder joints in the late periods of life.

BURSAL SIDE OF THE GLENO-HUMERAL JOINT

One hundred shoulder joints obtained from cadavers ranging in age from 19 to 88 years furnished the material for this investigation. The progressive rise in severity

of degenerative lesions observed in this study paralleled the alterations recorded above for the glenoid side of the cuff

SUBACROMIAL BURSA

In infant shoulders the roof and the lateral walls of the subacromial bursa consisted of a filmy transparent delicate membrane. Occasionally a fold was demonstrable separating the subacromial portion of the bursa from the subcoracoid portion. Specimens of later age periods in which there was evidence of advanced degenerative changes in the cuff exhibited thickened bursal walls and not infrequently the bursa was divided into several separate cavities by thick smooth adhesions. In all instances of a complete rupture of the cuff (in which there was direct communication between the joint and the subacromial bursa) the bursa was markedly enlarged in all its dimensions and its walls were hypertrophied and thickened. In fact in shoulder joints with complete avulsion of the rotator cuff the humeral head lay under the roof of the

subacromial bursa. It is reasonable to think that the increase in the size of the bursa in these instances is in response to the presence of synovial fluid which constantly distends the bursal sac during abduction and elevation of the arm.

It is an accepted clinical fact that frozen shoulders never are encountered in patients with complete tears of the cuffs. One may assume, therefore, that the presence of synovial fluid within the bursa prohibits the formation of an adhesive type of bursitis or tendinitis which may initiate pathologic processes responsible for frozen shoulder.

In a few shoulder joints of aged individuals the bursa was completely obliterated. There was discernible no interval between the subdeltoid fascia and the outer surface of the rotator cuff. Sharp dissection was necessary to separate the deltoid muscle from the outer surface of the musculotendinous cuff. All these specimens disclosed rotator cuffs which were intact but grossly degenerated.

FLOOR OF THE BURSA AND THE OUTER SURFACE OF THE ROTATOR CUFF

The intimate association of the floor of the subacromial bursa with the outer surface of the rotator cuff, makes it necessary to discuss these two structures as a unit.

Normally the base of the bursa is firmly adherent to the upper and outer portion of the greater tuberosity and to the rotator cuff where it inserts into the tuberosities. Its base bridges the bicipital sulcus. In infant shoulders and those of early decades the floor of the bursa was smooth, glistening and transparent. Palpation from within the bursa revealed that the tuberosities and the edge of the acromion were smooth and regular. Upon abduction and elevation of the humerus, the base of the bursa passed beneath the coraco-acromial ligament and the acromion without impingement.

Several specimens of the fifth decade exhibited gross manifestations of degenerative changes which increased in frequency

and severity in each subsequent decennium. Impairment of the base of the bursa depended upon the degree of the alterations in the underlying rotator cuff. Thickening of the synovial lining, villous formations and tabs were the lesions encountered most frequently. As a rule, they were associated with thinning, tearing, shredding and lamination of the rotator cuff fibers particularly in the supraspinatus and the infraspinatus regions (Fig. 88). In one specimen, the entire bursa was packed with numerous filmy, branching synovial villi arising from the base of the bursa. A small complete tear was also observed in the supraspinatus tendon.

As pointed out by Codman, concentration of bursal lesions in the supraspinatus region leads one to believe that this is an area of greater stress and strain and, upon elevation of the extremity, more likely to impinge against the coraco-acromial ligament and the acromion than are the remaining portions of the cuff.

Macroscopic calcareous deposits never were observed within the bursa. However in two specimens numerous small and discrete yellowish calcareous nodules were encountered beneath the floor of the bursa. They lay within the fibers of the supraspinatus and the infraspinatus tendons just proximal to their line of insertion into the facets of the greater tuberosity (Fig. 89, left). Another specimen exhibited one large round deposit measuring one quarter of an inch in diameter and one eighth of an inch high in the supraspinatus area. The floor of the bursa was stretched tightly over it and, upon abduction and elevation of the humerus the nodule impinged against the edge of the coraco-acromial ligament and the acromion (Fig. 89, right). It became obvious that such lesions may give rise to symptoms by irritation of the floor of the bursa and also by acting as a mechanical obstacle to free motion in abduction and elevation of the extremity.

Though not common, microscopic deposits of calcium were encountered more

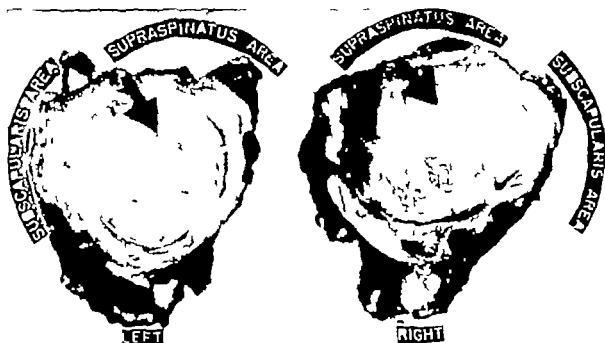


FIG. 88 Paired specimens from an individual 48 years of age. Arrows point to the irregularities in the floor of the subacromial bursa. This is a view looking directly on top of the bursa; the roof of the bursa has been excised in both specimens. Degenerative areas are observed at points making greater contact with the acromion.

noted that failure of the rotator muscles to stabilize the humeral head against the glenoid cavity during abduction causes an upward thrust of the head against the inferior surface of the acromion, thereby pinching and compressing the intervening soft tissue structures. Moreover, the rotator apparatus functions as the short arm of a long lever. This predisposes the musculotendinous cuff to repeated strain. Strains are produced also by various occupations which demand that the arm function in slightly abducted positions. The accumulative effect of slight, repeated traumata over many years is partially responsible for the advanced degenerative and attritional changes in capsular tissues of the shoulder joints in the late periods of life.

BURSAL SIDE OF THE GLENO-HUMERAL JOINT

One hundred shoulder joints obtained from cadavers, ranging in age from 19 to 88 years, furnished the material for this investigation. The progressive rise in severity

of degenerative lesions observed in this study paralleled the alterations recorded above for the glenoid side of the cuff.

SUBACROMIAL BURSA

In infant shoulders the roof and the lateral walls of the subacromial bursa consisted of a filmy, transparent delicate membrane. Occasionally a fold was demonstrable separating the subacromial portion of the bursa from the subcoracoid portion. Specimens of later age periods in which there was evidence of advanced degenerative changes in the cuff exhibited thickened bursal walls and not infrequently the bursa was divided into several separate cavities by thick smooth adhesions. In all instances of a complete rupture of the cuff (in which there was direct communication between the joint and the subacromial bursa) the bursa was markedly enlarged in all its dimensions and its walls were hypertrophied and thickened. In fact in shoulder joints with complete avulsion of the rotator cuff the humeral head lay under the roof of the

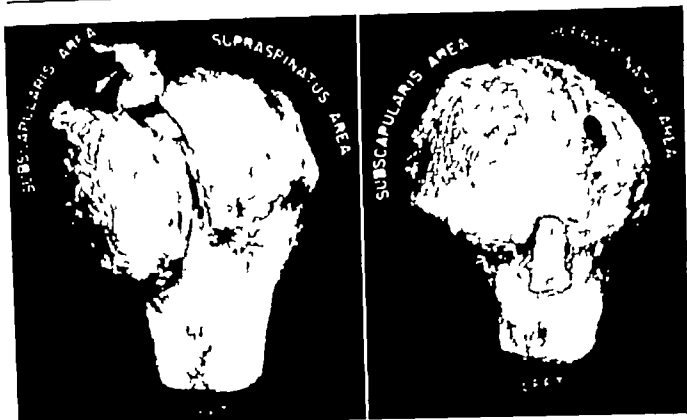


FIG 91 (Top left) Small longitudinal tear at junction of supraspinatus and infraspinatus tendons. Biceps tendon has been reflected upward and its inner edge is frayed. (Top right) Small complete tear in supraspinatus area of cuff Note slight elongation of circular defect (Bottom) Same features as above

Macroscopic degenerative changes were first noted in the cuff in specimens of the fourth period of life. Essentially, the lesions comprised thinning shredding lamination and tears of varying degree of the cuff. Although abnormalities were observed more frequently in the subscapularis region of the inner side of the cuff they were encountered more frequently in the supraspinatus area of the outer side of the cuff. Thinning of the tendon proximal to its line of insertion into the facet of the tuberosity was the lesion encountered most frequently. In some specimens the continuity of the cuff was maintained only by a thin ribbon of tissue. In many specimens the cuff on the inner side was thickened and hypertrophied proximal to this thin band. Such a hyperplastic process was nature's attempt to repair the defect. All the defects on the

synovial side of the cuff as previously noted were limited by a thickened synovial falciform edge also the result of a reparative process. In this series 13 complete tears of varying degree were observed (13 per cent). Figures 91 to 99 show the tears encountered. The first complete tear was found in the fifth decennium.

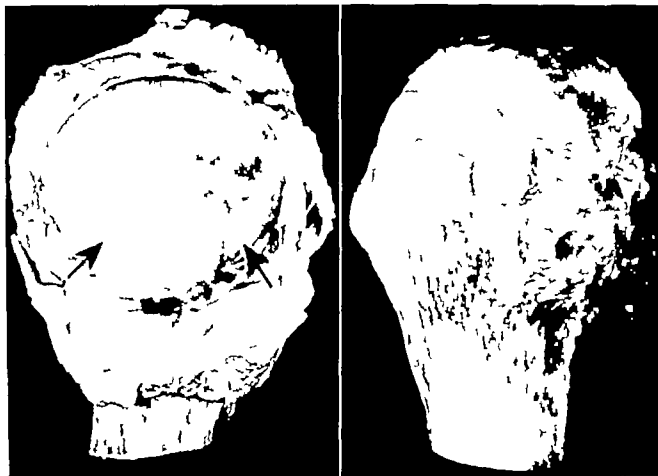


FIG 89 (*Left*) Arrows point to calcareous deposits within the musculotendinous cuff. The floor of the bursa is stretched tightly over the calcareous areas. (*Right*) Large calcareous deposit in supraspinatus portion of musculotendinous cuff. On abduction of the humerus this mass impinged against the acromion and the acromioclavicular ligament.



FIG 90 Well formed bone tissue appears in the supraspinatus tendon. The patient is 52 years of age and had no pain or shoulder dysfunction. ($\times 66$)

frequently than macroscopic deposits. It was significant that calcium deposits always were associated with gross evidence of degenerative changes in the musculotendinous cuff. This observation has been recorded by many observers and forces one to conclude that such lesions in this region are manifestations of degenerative alterations.

Oseous tissue in the cuff was disclosed in three specimens (Fig 90). It was observed in the supraspinatus tendon in 2 specimens; in the third the tendons of the supraspinatus, the infraspinatus and the teres minor muscles were converted into a continuous shell of dense bone.

The rotator tendons revealed a diversity of pathologic alterations which increased in severity in each subsequent decennium.

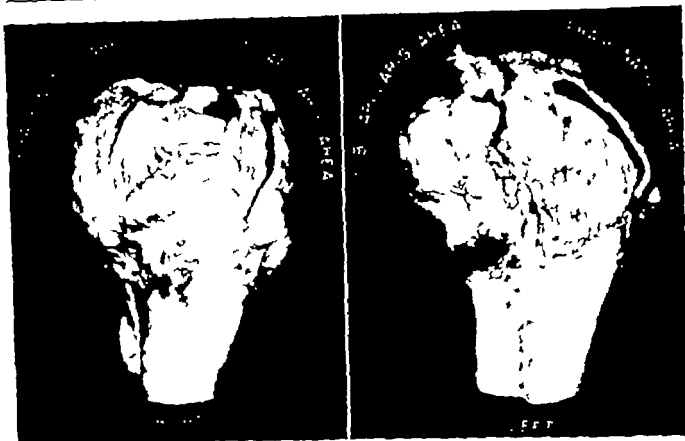


FIG 93 (Left) Advanced tearing away of fibers from the humeral head and longitudinal splitting of the cuff. Note secondary split in infraspinatus area of the cuff, converting this portion of remaining cuff into a ribbon. Atrophy and recession of the tuberosities are pronounced. The tear crossed the groove to involve the upper portion of the subscapularis tendon. The biceps tendon exhibits severe shredding, thickening and hypertrophy.

FIG 94 (Right) Elongation of tear is very evident approximating a triangular defect. Its edges are smooth and thin. The tuberosities are atrophic and disclose pronounced roughening. The biceps tendon is frayed, shredded and thin. No vestige of the distal end of the cuff remains. The defect involves both the infraspinatus and the supraspinatus areas.

by Codman) palpable to the examiner and the patient in shoulders with a partial or complete tear of the rotator cuff.

Other specimens revealed pronounced atrophy of the cuff proximal to the thinned portion inserted into the tuberosity. This atrophy may be a later stage of the hyperplastic process described previously. It appeared as if the reparative processes had ceased and most of the hypertrophied shredded ends of the torn fibers had been worn away (Figs 97, 98 and 99). In such instances the damaged portion of the cuff passed under the coraco-acromial arch without interference. In patients with partial or complete tears disappearance of pain may be accounted for by the removal of obstruction to free motion.

Shredding and fraying of the fibers in the supraspinatus and the infraspinatus regions of the cuff was observed in many specimens. Occasionally a depression in the cuff was discernible just proximal to its insertion into the humeral head (Fig 100). Alterations were most pronounced in and adjacent to this depression. Thus, one was forced to assume that this area firmly abutted against the coraco-acromial ligament or the acromion when the arm was abducted or elevated. Repeated trauma of this nature undoubtedly played a major role in the production of the alterations noted in the cuff.

A few specimens disclosed lamination of both the deep and the superficial layers of the musculotendinous cuff parallel with the

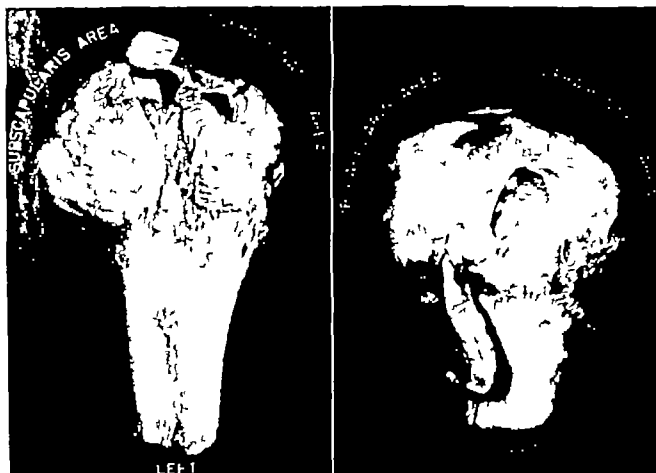


FIG 92 (Top left) Elongation of defect due to pull of fibers in direction of arrows. Bicapital groove has been opened for inspection. Note the well formed supratubercular ridge over which the biceps tendon plays. (Top right) Slightly larger complete tear in supraspinatus region of cuff. Edges are sharp smooth and thin. Note how the defect tends to become elongated in the direction parallel with the muscle fibers. (Bottom) Small complete tear which still shows little evidence of elongation.

Elevation and abduction of the humerus of these shoulders disclosed an interesting observation which may have some bearing on the symptomatology of partial ruptures of the rotator cuff. As the thickened portion of the cuff passed under the coraco-acromial ligament or the acromion a feeling of increased resistance and friction was noted and the thinned portion of the cuff buckled upon itself. It is reasonable to assume that, as the hypertrophied portion of the cuff passes under the acromion or the coraco-acromial ligament the patient experiences varying degrees of pain dependent upon the amount of friction produced. This pathologic alteration may be responsible also for the "jog" and the crepitus (first recorded



FIG 97 (*Left*) Advanced atrophy of the greater tuberosity. There is a large defect in the head of the humerus. The bone around the defect is eburnated and polished. There is an extensive cuff tear involving the supraspinatus and the infraspinatus areas. The edges of the tear are thin and smooth.

FIG 98 (*Right*) Large complete tear extending across the bicipital groove involving the subscapularis tendon. The biceps tendon is exposed and is frayed and thickened. Tuberosities show pronounced recession and atrophy.

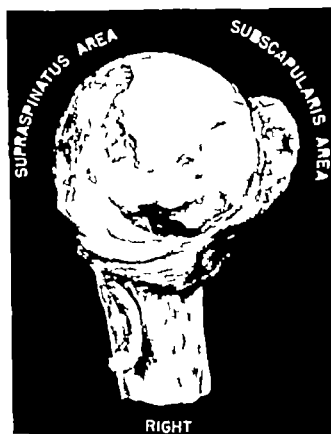


FIG 99 Massive avulsion of the cuff with recession of the tuberosities, erosion of the articular surface of the head of the humerus and formation of excrescences. Only a few fibers of the subscapularis tendon remain intact. The end of extracapsular portion of the biceps tendon has attained a bony attachment below the lesser tuberosity.

supraspinatus muscle pulls the proximal torn fibers medially thereby producing an equilateral triangular defect (Figs 92, *top left* 94 and 96). These observations support McLaughlin's concept of the mechanism of the production of ruptures with retraction of the cuff, but they fail to substantiate Codman's belief that the lateral ends of transverse tears are anchored while the center fibers retract medially.

Larger lesions usually involved the entire width of the supraspinatus area and extended for varying distances into the infraspinatus portion of the cuff (Figs 94 and 96). Roughly the defect resembled an

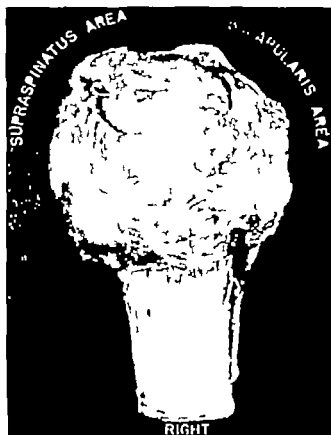


FIG 95 (Left) An unusual type of complete tear completely circular and involving all the supraspinatus area and a part of infraspinatus area of the cuff. Through the defect in the cuff can be seen the roughened pitted and irregular articular surface of the head of the humerus.

FIG 96 (Right) A triangular defect involving the supraspinatus and the infraspinatus regions of the cuff.

joint cavity. Fraying and shredding of the fibers were concomitant lesions in these specimens.

Complete tears of the cuff varied in extent from small almost imperceptible openings to avulsion of the entire cuff (Figs 91 to 99). Thirteen specimens in this series revealed complete tears of some degree. The supraspinatus area was involved in 46.1 per cent, the supraspinatus and the infraspinatus areas, in 38.4 per cent, the infraspinatus area alone in 0.7 per cent, and the entire cuff in 0.7 per cent. The subscapularis area alone never was completely torn. In 3 specimens it was implicated with tears in the supraspinatus and the infraspinatus tendons. Small ruptures in the supraspinatus area of the cuff were located just proximal to the line of insertion of the tendon fibers into the tuberosity.

Small tears failed to exhibit a specific configuration. In general they were more or less irregular circular defects with scarred hypertrophied or thinned peripheral margins. However many revealed a small longitudinal continuation of the defect parallel with the tendon fibers extending medially. This longitudinal rent usually was observed arising from the anterior aspect of the defect. Larger tears disclosed a more pronounced longitudinal rent making it clear that once a portion of the entire thickness of the cuff had pulled away at some point from its bony insertion it was subjected to opposing mechanical forces which split its fibers longitudinally.

The infraspinatus and the teres minor muscles pull the posterior margin backward while the subscapularis muscle pulls the anterior margin forward at the same time the

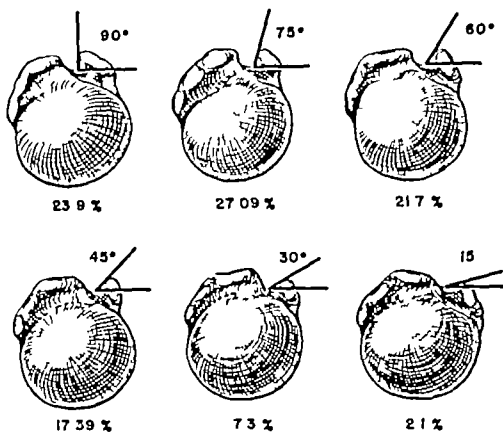


FIG 103 Drawing showing six variations and their incidences of the angle of medial wall of the intertubercular groove. (Redrawn with modification from Hitchcock and Bechtol J Bone & Joint Surg 30-A 263 273)

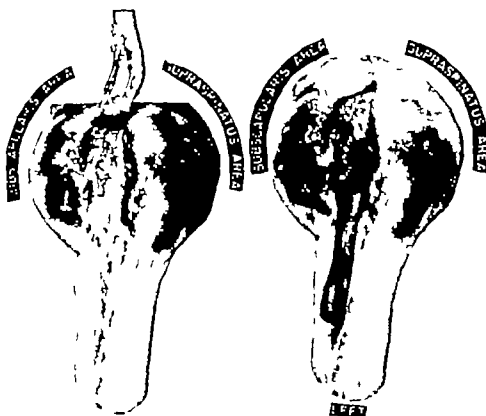


FIG 104 (Left) Note defect in frayed biceps tendon and spur in the floor of the bicipital groove. (Right) When the tendon is in its normal position the defect in its substance fits snugly around the bony spur



FIG 100 Note flattened roughened surface of the musculotendinous cuff over the greater tuberosity. This area impinged against the acromion upon abduction of the arm. A marker has been passed beneath a defect in the attachment of the subscapularis tendon. The tissues above the marker form a fascial sling for the biceps tendon which no longer lies in the intertubercular sulcus.



FIG 101 Undersurface of acromion process of specimen shown in Figure 99. The peripheral margin reveals advanced new bone formation. The undersurface of the acromion is eburnated and highly polished, resulting from constant contact with articular surface of the head of the humerus.



FIG 102 Schematic drawing showing a supratubercular ridge which may facilitate displacement of biceps tendon out of the intertubercular sulcus.

equilateral triangle with its base on the tuberosity. No vestige of the distal end of the cuff was demonstrable in these specimens. Bony excrescences over the tuberosities were encountered frequently. In some it became apparent that these hypertrophic lesions impinged upon the acromion during abduction of the arm. Evidence of such trauma was noted on the edge of the acromion by the presence of both bony spurs and roughening.

Several specimens with large defects in the cuff revealed advanced recession and atrophy of the tuberosities as if they were being leveled off gradually (Figs 93, 94, 97, 98 and 99). Two specimens revealed that the tear extended across the bicipital groove and involved the upper half of the subscapularis tendon (Figs 93 and 98). Another disclosed a massive avulsion of the cuff (Fig 99). The humeral head which showed advanced hypertrophic changes lay beneath the acromion which exhibited a dense

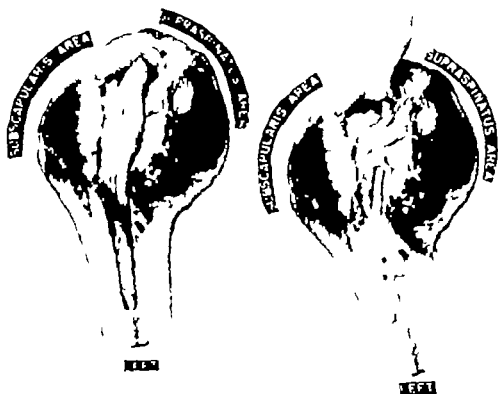


FIG 105 (Left) Biceps tendon lies to inside of the lesser tuberosity (Right) Note the reduction in the height of the lesser tuberosity also a well formed fascial sling extending from the fibrous capsule to the remainder of the lesser tuberosity The undersurface of the biceps tendon reveals advanced degenerative changes.

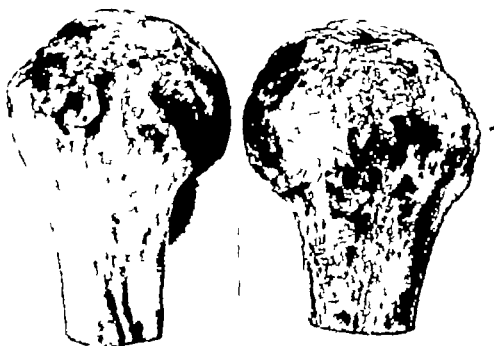


FIG 106 (Left) Well formed bony excrescences over both tuberosities. The intertubercular sulcus is narrowed and the head of the humerus proximal to the sulcus is flattened and eburnated The biceps tendon of this specimen was thickened frayed and shredded (Right) Note complete atrophy and recession of tuberosities obliteration of intertubercular sulcus, excrescences in bicipital groove and eburnation of the head of the humerus, anterior superior and posterior to the bicipital groove. The biceps tendon was thinned frayed and shredded

eburnated and highly polished inferior surface (Fig 101)

TUBEROSITIES AND BICIPITAL GROOVE

Irregular new bone formation, in varying degrees, over the tips of the tuberosities was the most outstanding pathologic alteration discernible in specimens after the fifth decade. This, together with thinning or complete rupture of the cuff in the supraspinatus area, was responsible for the formation of a depression (sometimes palpable through the deltoid) in the floor of the subacromial bursa. Codman pointed out

The tuberosity forms a distinct eminence and may cause a jog and friction as it passes upward under the acromion. The result is a stimulation of the bone cells in the tuberosity and irregular excrescences formed at the region where undue friction occurs. This new formed bone is very spongy and probably later atrophies since one finds that complete recession of the whole tuberosity has taken place in the very old cases.

Atrophy of the tendon without actual rupture might cause enough irregularity in the base of the bursa to begin a little irritation. A vicious circle of atrophy of tendon in inflammation of tuberosity, osteitis, excrescences, greater jog and greater friction, greater atrophy, etc., might be established. At the same time hypertrophic changes in the acromial groove are stimulated.

There is complete agreement with this cycle of events and subsequent changes in the tuberosities.

As the result of these bony lesions, alterations in the configuration of the bicipital groove were demonstrable. Not infrequently in specimens exhibiting marked hypertrophic new bone formation over and around the tuberosities the bicipital sulcus was narrowed and occasionally obliterated by these lesions. Bony spurs often were observed on the inner and the outer surfaces of the bicipital sulcus, being more common in the intertubercular portion of the groove. It becomes apparent that such alterations necessarily must interfere with normal excursion of the humeral head on the biceps tendon. This fact was substantiated by the

severe degenerative changes observed in the biceps tendons (Fig 104).

In specimens with marked atrophy and recession of the tuberosities, the intertubercular portion of the bicipital sulcus was worn away completely in many instances. The biceps tendon, which had become displaced from the sulcus, now occupied a position on the inner aspect of the remains of the lesser tuberosity. It was supported by a fascial sling such as described by Meyer (Fig 105). Six of the specimens studied in the first part of this chapter and 4 of those studied in the second disclosed complete absence of the intracapsular portions of the biceps tendons. The extracapsular ends were found attached to the humeral shafts below the lesser tuberosities (Fig 133).

In this investigation the bicipital groove exhibited many significant variations. Meyer (1928) first described a supratubercular ridge which is a ridge of bone continuous with the medial wall of the groove and extends proximally toward the articular margin of the humeral head (Fig 102). Its size and configuration vary considerably. In this study it was a pronounced structure in 73.8 per cent of the humeri, moderately developed in 31.5 per cent and absent in 43.4 per cent. Hitchcock and Bechtol found the ridge markedly developed in 8 per cent, moderately developed in 59 per cent and absent in 33 per cent. This supratubercular ridge, as pointed out by Meyer (1928), favors dislocation of the bicipital tendon by levering it out of the proximal portion of the groove. Supratubercular ridges, sufficiently high to displace the tendon against the transverse humeral ligament, are responsible for repeated trauma inflicted to the tendon and the tendon sheath during joint motion. Hitchcock and Bechtol (1938) noted that the height of the groove depended directly upon the obliquity of its medial wall. They found the angle of the medial wall to be 90° in 10 per cent of their humeri, 75° in 35 per cent, 60° in 34 per cent, 45° in 13 per cent, 30° in 6 per cent and 15° in 2 per cent. This investigation

revealed the angle of the medial wall to be 90° in 23.9 per cent, 75° in 27.09 per cent, 60° in 21.7 per cent, 45° in 7.3 per cent, 30° in 7.3 per cent, and 15° in 2.1 per cent (Fig 103). It was further observed that the base of the supratubercular ridge also was a factor in diminishing the depth of the groove. Nine per cent of the humeri with a supratubercular ridge disclosed this variation. In these specimens, the base of the ridge was unusually broad and continued across the floor of the groove toward its lateral wall (Fig 102). Bony spurs were observed on the medial wall in 9 per cent of the specimens, on the lateral wall in 13 per cent, and on both walls in 7.3 per cent. Spur formation undoubtedly follows inflammatory changes in the groove; such bony structures render the biceps tendon vulnerable to repeated trauma and attritional alterations (Figs 104, 106, 107, *right* and 108 *right*).

It was interesting to note that the height of the lesser tuberosity was reduced markedly in many specimens of the later decades.

In the light of the observations recorded in this study, it becomes apparent that physiologic aging plays a major role in the production of degenerative alterations in the musculotendinous cuff. However, such factors as occupation, excessive usage, debilitating disease, and repeated minor trauma in a measure also must be considered as responsible. Study of the cases in the second part of this chapter has revealed that many of the above-recorded lesions, particularly partial ruptures of the cuff (rim rents) and complete ruptures in some instances, are compatible with normal joint function without pain. We are forced to believe that symptoms and disability often are not caused by the lesion *per se* but by the associated sequelae which interfere with normal mechanics of shoulder joint function.

SUMMARY

1 Progressive degenerative alterations are noted in the articular cartilage of the

glenoid cavity as early as the second decade. In the early decades, these changes consisted of unevenness, fibrillations and furrows. Pitting erosions and marginal bone proliferations occurred later. The central depressed area in the center of the comma-shaped head we believed to be an area of greater contact than the surrounding cartilage. These progressive changes reached their height in the sixth decade.

2 The labrum glenoidale, because of its anatomic position surrounding the glenoid cavity and because of its intimate relationship to the biceps tendon and the synovial ligaments, is subjected to stress and strain during normal shoulder function. Hence degenerative changes manifest themselves rather early and become more pronounced and frequent with each subsequent decade. These alterations consist of labral detachments, thinning, scalloping, undulations and synovial tabs and fringes. Labral detachments may be interpreted erroneously as the result of local trauma and as the underlying pathology of recurrent dislocations of the shoulder. The above findings fail to substantiate Bankart's contention that labral detachment is the causative factor in producing recurrent dislocations of the shoulder, and also provide an explanation for the frequent failures of the various suspension operations (such as the Nicola procedure) utilized to stabilize recurrent dislocating shoulders.

3 No significant changes were noted relative to the biceps tendon until the fifth decade. These consisted of thickening, widening, and shredding, becoming more pronounced with each subsequent age group. It is of interest to note that these changes occurred concurrently with the appearance of degenerative lesions in the musculotendinous cuff. It is reasonable to assume, therefore, that as degenerative lesions begin to manifest themselves in the musculotendinous capsule, the biceps tendon is forced to function as one of the main supports of the upper extremity. Hence it is subjected to great attritional forces which

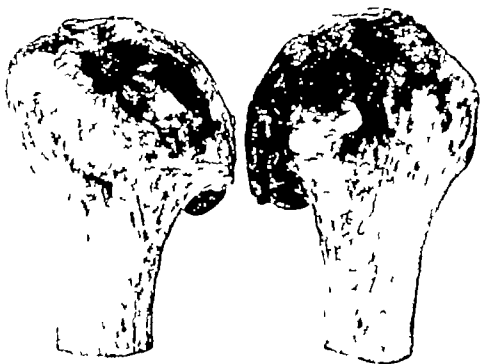


FIG 107 (*Left*) Posterior view A large defect in the postero-lateral aspect of the head of the humerus with formation of numerous excrescences around the defect. (*Right*) Anterior view Intertubercular groove is narrowed by excrescences. In this instance the biceps tendon was frayed and attenuated. Note profound degenerative changes and roughened bone in humeral head proximal to the intertubercular groove Also osseous and cartilaginous changes due to an old compression fracture through the anatomic neck of the humerus.



FIG 108 (*Left*) Posterior aspect Complete atrophy of the greater tuberosity and new bone formation on the posterior aspect of the shaft of the humerus below the level of the neck. (*Right*) Anterior aspect. Bicipital groove is completely obliterated by new bone formation. In this specimen no intracapsular portion of the biceps tendon was found. The proximal end of the extracapsular portion was buried in the osseous tissue obliterating the groove.

revealed the angle of the medial wall to be 90° in 23.9 per cent, 75° in 27.09 per cent, 60° in 21.7 per cent, 45° in 17.3 per cent, 30° in 7.3 per cent, and 15° in 2.1 per cent (Fig. 103). It was further observed that the base of the supratubercular ridge also was a factor in diminishing the depth of the groove. Nine per cent of the humeri with a supratubercular ridge disclosed this variation. In these specimens, the base of the ridge was unusually broad and continued across the floor of the groove toward its lateral wall (Fig. 102). Bony spurs were observed on the medial wall in 9 per cent of the specimens, on the lateral wall in 13 per cent and on both walls in 7.3 per cent. Spur formation undoubtedly follows inflammatory changes in the groove; such bony structures render the biceps tendon vulnerable to repeated trauma and attritional alterations (Figs. 104, 106, 107, *right* and 108 *right*).

It was interesting to note that the height of the lesser tuberosity was reduced markedly in many specimens of the later decades.

In the light of the observations recorded in this study it becomes apparent that physiologic aging plays a major role in the production of degenerative alterations in the musculotendinous cuff. However, such factors as occupation, excessive usage, debilitating disease and repeated minor trauma, in a measure, also must be considered as responsible. Study of the cases in the second part of this chapter has revealed that many of the above recorded lesions, particularly partial ruptures of the cuff (rim rents) and complete ruptures in some instances, are compatible with normal joint function without pain. We are forced to believe that symptoms and disability often are not caused by the lesion *per se* but by the associated sequelae which interfere with normal mechanics of shoulder joint function.

SUMMARY

1. Progressive degenerative alterations are noted in the articular cartilage of the

glenoid cavity as early as the second decade. In the early decades, these changes consisted of unevenness, fibrillations and furrows. Pitting, erosions and marginal bone proliferations occurred later. The central depressed area in the center of the comma's head we believed to be an area of greater contact than the surrounding cartilage. These progressive changes reached their height in the sixth decade.

2. The labrum glenoidale, because of its anatomic position surrounding the glenoid cavity and because of its intimate relationship to the biceps tendon and the synovial ligaments, is subjected to stress and strain during normal shoulder function. Hence, degenerative changes manifest themselves rather early and become more pronounced and frequent with each subsequent decade. These alterations consist of labral detachments, thinning, scalloping, undulations and synovial tabs and fringes. Labral detachments may be interpreted erroneously as the result of local trauma and as the underlying pathology of recurrent dislocations of the shoulder. The above findings fail to substantiate Bankart's contention that labral detachment is the causative factor in producing recurrent dislocations of the shoulder, and also provide an explanation for the frequent failures of the various suspension operations (such as the Nicola procedure) utilized to stabilize recurrent dislocating shoulders.

3. No significant changes were noted relative to the biceps tendon until the fifth decade. These consisted of thickening, widening, and shredding, becoming more pronounced with each subsequent age group. It is of interest to note that these changes occurred concurrently with the appearance of degenerative lesions in the musculotendinous cuff. It is reasonable to assume, therefore, that as degenerative lesions begin to manifest themselves in the musculotendinous capsule, the biceps tendon is forced to function as one of the main supports of the upper extremity. Hence, it is subjected to great attritional forces which



FIG 109 D. A. male white age 43
Right shoulder A finished specimen
which has been mounted on a circular
glass rod frame so that the synovial side
of the musculotendinous cuff the biceps
tendon and the articular surfaces of the
head of the humerus are clearly visual-
ized.

are responsible for the various alterations in the tendon

4 Variations as to size location and number of the synovial ligaments and synovial recesses are numerous These variations may be developmental the result of synovial changes in the subscapularis area or alterations in the biceps tendon and the labrum The clinical significance of these findings is obvious when a shoulder joint is explored to correct the causative pathology responsible for recurrent dislocations. One may readily misinterpret a large synovial recess for a rent in the capsule and a middle glenohumeral ligament as a detached and displaced labrum

5 Changes in the musculotendinous capsule first appeared in the fifth decade These changes varied from minor incomplete tears to complete tears in one or all of the rotator

tendons In the presence of a tear the synovial lining formed a falciform ligament across the defect as if attempting to repair it. The study of the inside of the glenohumeral joint revealed 24 per cent supraspinatus tears and 41 per cent subscapularis tears, while that on the outside of the cuff disclosed complete tears in only 13 per cent of the specimens Such lesions were more common and of greater severity in the later decenniums, leading one to believe that they are degenerative lesions due to wear and tear and to aging"

6 Lesions of the inside of the musculotendinous cuff are encountered more frequently and of greater severity than those on the outside of the cuff

7 The biceps tendon exhibits degenerative alterations which progress in gradient in each successive decade, beginning with the fifth. These lesions are associated with aging and also are caused by the changes in the bony configuration of the bicipital groove produced by excrescences spurs and new bone formation.

LESIONS OF THE SHOULDER COMPATIBLE WITH GOOD FUNCTION

INTRODUCTION

The investigation recorded in the first part of this chapter brought to light many significant observations relative to the degenerative lesions manifested in the biceps tendon the labrum glenoidale and the musculotendinous cuff in different age groups Unfortunately the gross study was conducted on shoulder joints of individuals on whom no histories were available prior to death while the microscopic study was made on shoulders obtained from individuals on whom histories were obtained and examinations made of the shoulders prior to death, but no attempt was made to correlate these clinical data with the observations noted other than that these patients had sustained no severe injuries during their lifetime Therefore one was unable to eval

uate the true clinical significance of these observations. However, it was felt that the findings were of sufficient importance to pursue the study further. Hence the following investigation was undertaken on a series of shoulder joints obtained from individuals postmortem on whom sufficient clinical data were obtained prior to death to permit correlation of the abnormalities noted in the component structures of the shoulder joint with the functional capacity of the joint. It was of great significance to observe the pronounced severity of degenerative lesions in

the musculotendinous cuffs and the biceps tendons that is compatible with good function. This study demonstrated further the sequence and the manner in which the degenerative alterations occurred. Finally, one was made aware that our present-day concept of the management of many lesions of the shoulder needs further investigation and revision.

MATERIALS AND METHODS

Ninety six shoulder joints were obtained postmortem from 50 individuals. A clinical

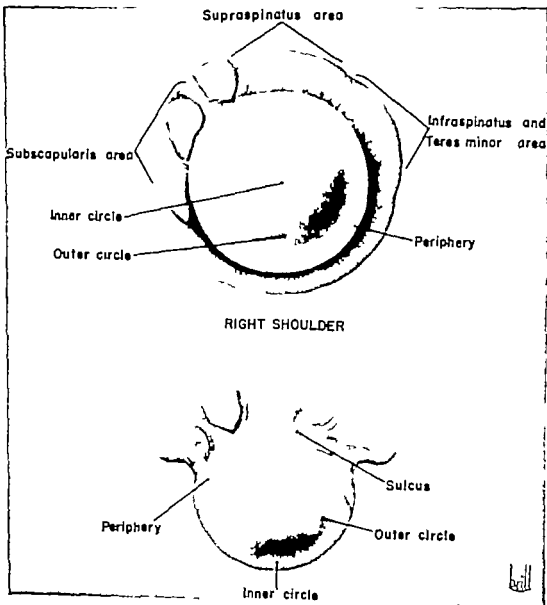


FIG. 110 Schematic drawing of finished mounted specimen showing division of articular cartilage into the inner circle, the outer circle, and periphery, the sulcus which is produced by recession of the cuff, and the designated areas of the musculotendinous cuff.



FIG 109 D. A. male white age 43 Right shoulder A finished specimen which has been mounted on a circular glass rod frame so that the synovial side of the musculotendinous cuff the biceps tendon and the articular surfaces of the head of the humerus are clearly visualized.

are responsible for the various alterations in the tendon.

4 Variations as to size location and number of the synovial ligaments and synovial recesses are numerous. These variations may be developmental the result of synovial changes in the subscapularis area or alterations in the biceps tendon and the labrum. The clinical significance of these findings is obvious when a shoulder joint is explored to correct the causative pathology responsible for recurrent dislocations. One may readily misinterpret a large synovial recess for a rent in the capsule and a middle glenohumeral ligament as a detached and displaced labrum.

5 Changes in the musculotendinous capsule first appeared in the fifth decade. These changes varied from minor incomplete tears to complete tears in one or all of the rotator

tendons. In the presence of a tear the synovial lining formed a falciform ligament across the defect as if attempting to repair it. The study of the inside of the glenohumeral joint revealed 24 per cent supraspinatus tears and 41 per cent subscapularis tears, while that on the outside of the cuff disclosed complete tears in only 13 per cent of the specimens. Such lesions were more common and of greater severity in the later decenniums, leading one to believe that they are degenerative lesions due to wear and tear and to aging."

6 Lesions of the inside of the musculotendinous cuff are encountered more frequently and of greater severity than those on the outside of the cuff.

7 The biceps tendon exhibits degenerative alterations which progress in gradient in each successive decade, beginning with the fifth. These lesions are associated with aging and also are caused by the changes in the bony configuration of the bicipital groove produced by excrescences, spurs and new bone formation.

LESIONS OF THE SHOULDER COMPATIBLE WITH GOOD FUNCTION

INTRODUCTION

The investigation recorded in the first part of this chapter brought to light many significant observations relative to the degenerative lesions manifested in the biceps tendon the labrum glenoidale and the musculotendinous cuff in different age groups. Unfortunately the gross study was conducted on shoulder joints of individuals on whom no histories were available prior to death while the microscopic study was made on shoulders obtained from individuals on whom histories were obtained and examinations made of the shoulders prior to death but no attempt was made to correlate these clinical data with the observations noted other than that these patients had sustained no severe injuries during their lifetime. Therefore one was unable to eval

history was obtained and an examination of the shoulders made and recorded on these individuals prior to their deaths. Only shoulder joints of patients who believed that they possessed normal shoulders, who were unaware of any disability and disclosed no shoulder dysfunction by examination were selected for this study. At this place it must be pointed out that the above clinical data were obtained from individuals who were mentally alert and competent to give accurate information. Many of these patients were kept under observation for many weeks before death occurred. Moreover, the patients who provided the specimens for study formed a small group of the patients examined for this investigation because many of the individuals studied were fortunate enough to recover from their malady and to be discharged from the hospital. In only two instances was it necessary to obtain the clinical data desired from relatives of the deceased. Most of these individuals gave a history that at some time in their lives they had some temporary disability of the shoulder; none, however, had any severe traumatic lesions to the shoulder followed by marked dysfunction. One gave a history which was consistent with that of a frozen shoulder on both sides (Fig. 133). Only 2 patients in this group had some pain on motion for 6 months prior to death. Movements in the shoulder, however, were not impaired. There were 36 males and 14 females; 48 right and 48 left shoulders; the age ranged from 18 to 74 years. Roentgenograms of all shoulders were made, the specimens were then mounted (Fig. 109) and studied.

For the purpose of study and presentation the articular surface of the head of the humerus was divided into three parts: the central area was designated the inner circle; the outer area, the outer circle; and the margin of the articular cartilage, the periphery. The interval between the periphery and the inner surface of the musculotendinous cuff was designated the sulcus, while the musculotendinous cuff was divided into



FIG. 113. R. C., female, white, age 52. Left shoulder. This is the only specimen of this series that revealed an erosion in the inner circle. Note the depression in the superior aspect of the head in that region of the periphery adjacent to the supraspinatus tendon. Also the extensive, incomplete tear which implicates all the supraspinatus and infraspinatus regions of the cuff. The humeral defect may be the result of a growth defect or unrecognized trauma.

the subscapularis, the supraspinatus and the infraspinatus and the teres minor areas (Fig. 110).

OBSERVATIONS REFERABLE TO THE ARTICULAR CARTILAGE OF THE HEAD OF THE HUMERUS

It was interesting to note that alterations in the articular cartilage of the head of the humerus were never extensive even in the very late decades of life. They did not parallel the changes observed in the articular cartilage of the glenoid cavities of representative specimens of the same age period (Fig. 111). For example, the humeral head in Figure 111, left, discloses only minimal changes when compared with the glenoid cavity of the same shoulder joint shown in Figure 111, right. As a rule, the

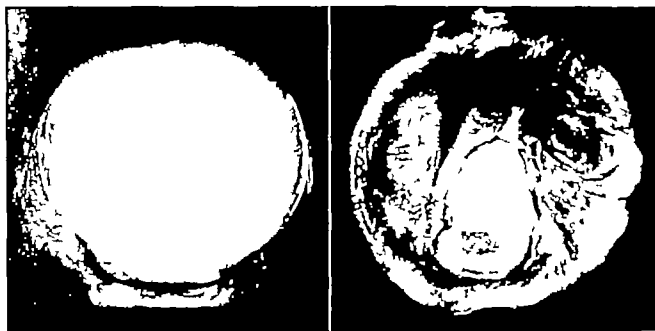


FIG 111 (*Left*) Articular surface of the head of the humerus exhibits only minimal degenerative alterations as compared with those noted in the glenoid fossa of the same shoulder (*right*) This was true of most of the shoulder joints in this series. (*Right*) Degenerative changes in the articular cartilage of the glenoid cavity are more pronounced than those in the head of the humerus of the same shoulder joint (*left*)

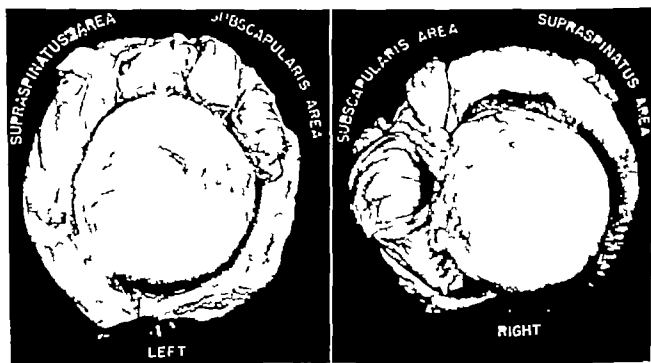


FIG 112 (*Left*) W S, male white age 67 Left shoulder Note the increase in gradient of degenerative lesions in the outer circle over those in the inner circle. This ratio was a constant feature in the great majority of shoulders exhibiting alterations in the articular cartilage of the head of the humerus. (*Right*) Right shoulder of the same patient. As in the illustration at the left there are noted more pronounced degenerative lesions in the outer circle and periphery of the articular surface of the head of the humerus than in the inner circle. Also observe the extensive incomplete tear in the subscapularis portions of the cuff and the recession of the torn cuff from the periphery of the articular margin.



FIG 118 (Left) D F male white age 55 Right shoulder There are advanced degenerative lesions implicating the whole synovial side of the musculotendinous cuff, particularly in the supraspinatus and infraspinatus areas. The cuff has receded a considerable distance from the margin of the articular cartilage forming a wide sulcus (Right) Same shoulder, viewing the cuff from the outside Note that it is impossible to evaluate the severity of the lesions on the inside of the cuff by the appearance of its outer surface

inner circle exhibited the least severe alterations while the most profound lesions were found along the periphery of the articular cartilage. The outer circle in most instances disclosed degenerative changes of greater severity than those noted in the articular cartilage of the inner circle. The alterations in the inner and the outer circles comprise thinning, fibrillation, pitting and erosion of the articular cartilage. Along the periphery the findings consist of pitting and erosions of the cartilage and formation of bony spurs and excrescences (Figs 112 and 113). Only one specimen of the series (a specimen in the sixth decade) disclosed a true erosion of the articular cartilage in the inner circle (Fig 113). One specimen (Fig 115) of the sixth decade exhibited maximum alterations in articular cartilage of the inner and the outer circles encountered in this series. Although there was a gradual rise in gradient of the degenerative lesions in each successive decade starting with the third the changes never reached such profound proportions as they did in the glenoid cavities nor did they parallel the extensive changes

noted in the musculotendinous cuff (Fig 116). This observation points out that development of degenerative changes in the soft tissue components of joints is not secondary to abnormalities in the cartilaginous and osseous tissues. During joint motion only a small portion of the articular surface of the large head of the humerus is in contact with that of the small glenoid cavity. It becomes apparent that contact areas in the glenoid cavity are subjected to more wear and tear than contact areas in the humeral head. This readily explains the discrepancies in the severity of the changes in the articular surfaces of the humeral head and glenoid fossa.

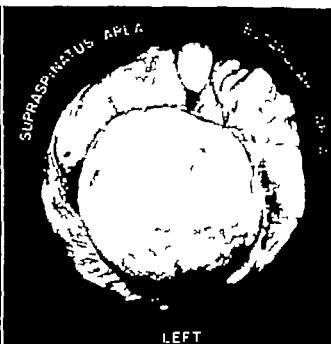
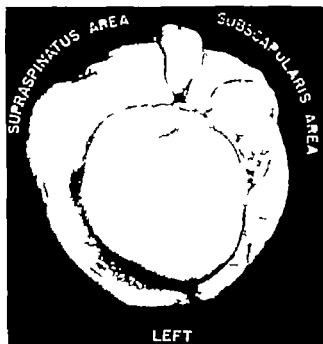


FIG 114 (Left) R G male white, age 32 Left shoulder Note indentation in superior portion of the head of the humerus also the irregularity of the entire head. The defect may be the result of unrecognized trauma or a growth defect.

FIG 115 (Right) J P., male white age 57 Left shoulder This specimen exhibits the most pronounced degenerative lesions in the articular cartilage of the head of the humerus. Those in the outer circle and the periphery disclose greater severity than those in the inner circle.

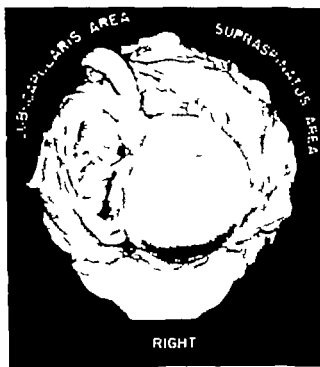


FIG 116 (Left) R. M. female white age 55 Right shoulder Degenerative changes in the articular cartilage of the head of the humerus do not parallel those in musculotendinous cuff Note in this specimen the severe alterations in the subscapularis regions of the cuff and only minimal lesions in the humeral head

FIG 117 (Right) D A. male white age 23 Left shoulder This specimen discloses no macroscopic evidence of degenerative changes. Note that the musculotendinous cuff with its synovial lining is in close proximity to the margin of the articular cartilage. Nowhere is there any recession of the cuff fibers.



FIG 118 (*Left*) D F male, white, age 55 Right shoulder There are advanced degenerative lesions implicating the whole synovial side of the musculotendinous cuff, particularly in the supraspinatus and infraspinatus areas. The cuff has receded a considerable distance from the margin of the articular cartilage, forming a wide sulcus. (*Right*) Same shoulder, viewing the cuff from the outside. Note that it is impossible to evaluate the severity of the lesions on the inside of the cuff by the appearance of its outer surface.

inner circle exhibited the least severe alterations, while the most profound lesions were found along the periphery of the articular cartilage. The outer circle in most instances disclosed degenerative changes of greater severity than those noted in the articular cartilage of the inner circle. The alterations in the inner and the outer circles comprise thinning fibrillation pitting and erosion of the articular cartilage. Along the periphery the findings consist of pitting and erosions of the cartilage and formation of bony spurs and excrescences (Figs 112 and 113). Only one specimen of the series (a specimen in the sixth decade) disclosed a true erosion of the articular cartilage in the inner circle (Fig 113). One specimen (Fig 115) of the sixth decade exhibited maximum alterations in articular cartilage of the inner and the outer circles encountered in this series. Although there was a gradual rise in gradient of the degenerative lesions in each successive decade starting with the third the changes never reached such profound proportions as they did in the glenoid cavities, nor did they parallel the extensive changes

noted in the musculotendinous cuff (Fig 116). This observation points out that development of degenerative changes in the soft tissue components of joints is not secondary to abnormalities in the cartilaginous and osseous tissues. During joint motion only a small portion of the articular surface of the large head of the humerus is in contact with that of the small glenoid cavity. It becomes apparent that contact areas in the glenoid cavity are subjected to more wear and tear than contact areas in the humeral head. This readily explains the discrepancies in the severity of the changes in the articular surfaces of the humeral head and glenoid fossa.

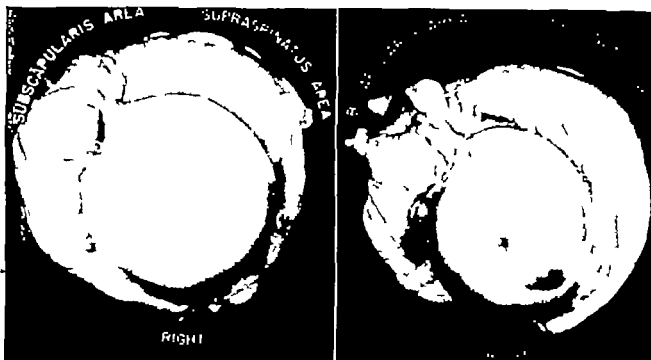


FIG 119 (Left) A. G, male, white, age 47 Right shoulder Large incomplete tear exists in the subscapularis tendon. Note the thickened synovial membrane distal to the defect in the cuff this is nature's method of trying to limit the progress of the tear, designated by Codman the falciform ligament.

FIG 120 (Right) J. M. male, white, age 54 Right shoulder A moderate size incomplete tear in the supraspinatus and infraspinatus areas of the cuff

SULCUS

In shoulders of infants and of individuals in the early decades the rotator cuff in the supraspinatus the subscapularis and the infraspinatus and the teres minor areas filled completely the interval between the tuberosities and the periphery of the articular cartilage. This is demonstrated in Figure 117 a specimen of the third decade. In the fourth decade several shoulders revealed a gradual tearing away of the cuff from the periphery. This change was observed more frequently in the supraspinatus area than the other portions of the musculotendinous cuff. Pitting bony spurs and excrescences were common findings in the sulcus. The afore mentioned alterations increased in intensity in each subsequent decade. In some specimens the periphery revealed subchondral new bone formation its articular edge was raised thickened irregular and studded with cartilaginous nodules in some instances. The degree of severity of the de-

generative lesions in the sulcus was in direct proportion to those in the adjacent musculotendinous cuff. As more and more fibers tore away the insertion of the cuff became thinner the width of the sulcus was dependent upon the extent of the recession of the cuff from its bony insertion (Fig 118 left)

SYNOVIAL MEMBRANE AND MUSCULOTENDINOUS CUFF

Macroscopic synovial alterations were first discernible in the second decade. They comprised shredding fraying tabs and villous formation. A gradual progression in severity was demonstrable in each successive age period however the lesions paralleled those noted in the musculotendinous cuff. Specimens of the sixth decade revealed lesions of the greatest severity in the synovial membrane.

Tears on the synovial side of the musculotendinous cuff were first observed in the



FIG 121 (Left) T C, male white, age 43 Right shoulder Large incomplete tear in supraspinatus area of the cuff Note the recession of the cuff fibers and thickening of the torn fibers. (Right) Left shoulder Large incomplete tear in supraspinatus and infraspinatus portion of the cuff, with recession of cuff and marked thickening of the torn fibers.

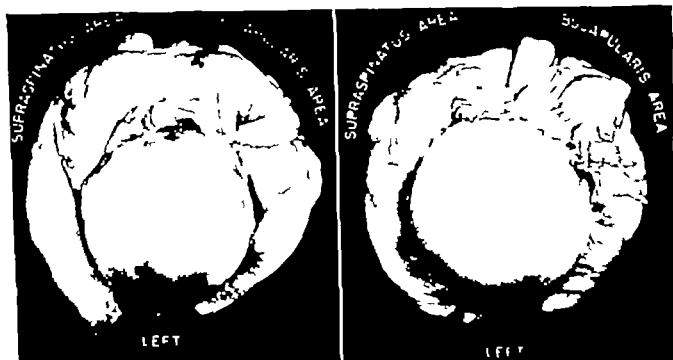


FIG 122 (Left) J M, male, white, age 54 Left shoulder Small incomplete tear in the supraspinatus region of the cuff

FIG 123 (Right) S R, male white, age 55 Left shoulder Advanced degenerative lesions of the synovial lining the musculotendinous cuff and incomplete tears of the subscapularis, the supraspinatus and the infraspinatus areas. Note the thickened, hyperplastic torn fibers and the minimal changes in the articular cartilage of the head of the humerus compared with the extensive alterations in the cuff

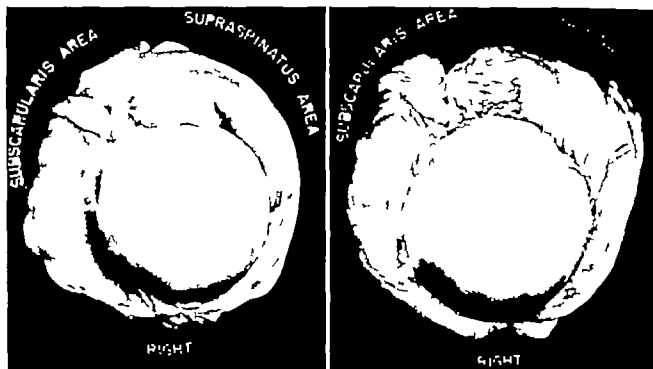


FIG 124 (Left) R. C female white age 62 Right shoulder Large incomplete tear exists in the supraspinatus and a small part of the infraspinatus regions of the cuff Note once more the marked hypertrophy of the torn fibers

FIG 125 (Right) J C., male colored age 54 Right shoulder There is a moderate incomplete tear in the supraspinatus region of the cuff and also considerable degenerative changes in the synovialis of this area and in the synovial membrane lining the subscapularis area of the cuff

fifth decennium Like other degenerative lesions these showed a gradual rise in gradient in each successive decade the most severe abnormalities were encountered in the sixth and the seventh decades In this series there were found 20 subscapularis tears (20.8 per cent) and 35 supraspinatus and infraspinatus tears (37.3 per cent) These findings differ from those noted in the discussion on the glenoid side of the glenohumeral joint in which the incidence of subscapularis tears was greater than that of supraspinatus tears. As a rule tears of the supraspinatus areas extended into the infraspinatus region of the cuff This feature is not appreciated when only the outer side of the cuff is inspected The appearance of the outer side of the musculotendinous cuff gives an erroneous impression of the extent of the degenerative lesions existing in the structure Only by visualizing the inside of the cuff can one determine the severity of

the changes (Fig 118) This is particularly true of complete tears of the cuff There were nine complete tears in this group of specimens

It was interesting to note the types and the extent of incomplete tears that are compatible with normal function they varied from small tears in and shredding of the cuff to extensive areas of tearing fraying and thinning of the subscapularis and the supraspinatus and infraspinatus insertion of the rotator cuff Pronounced thickening and hypertrophy were characteristic features of the torn cuff fibers (Figs. 119 to 128 inclusive)

Two shoulders (Fig 128) were obtained from a male 59 years of age This individual was unaware of any shoulder dysfunction at the time of examination except for some restriction of external rotation no other abnormalities were noted clinically in either shoulder

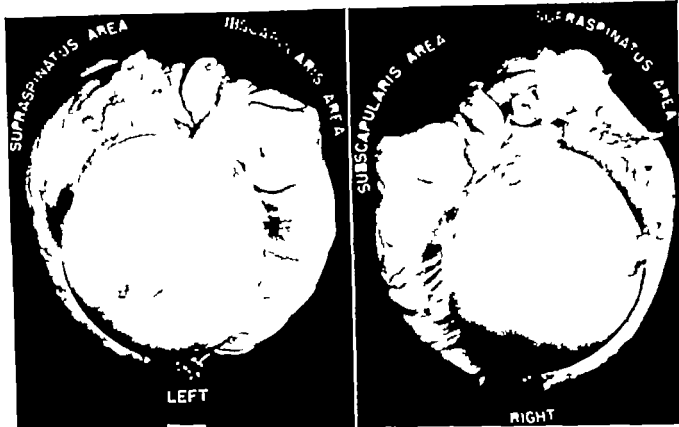


FIG 126 (Left) C D female colored age 56 Left shoulder Observe the marked fraying, tearing and shredding of the fibers of the musculotendinous cuff in the supraspinatus and part of infraspinatus regions of the cuff Again note the marked hypertrophy of the torn fibers. (Right) Right shoulder There is considerable tearing of the fibers of the entire supraspinatus and in infraspinatus regions of cuff, also marked hypertrophy of the torn fibers and recession of cuff from the periphery of the articular margin in the affected region.



FIG 127 J S male white age 54 Right shoulder An unusually large tear exists implicating the entire supraspinatus and a portion of the infraspinatus region of the cuff There is unusual hypertrophy and thickening of the torn fibers and marked recession and thinning of the remaining intact fibers of the cuff

The right shoulder (Fig 128 right) revealed advanced degenerative changes of the synovial membrane The alterations comprised tabs fraying hyperplasia and villous formation It exhibited an extensive incomplete tear in the musculotendinous cuff including all the supraspinatus and part of the infraspinatus tendon whereas the articular cartilage of the humeral head showed only minimal lesions

All the above changes were more pronounced in the left shoulder (Fig 128 left) The incomplete cuff tear extended from the middle of the infraspinatus tendon to the

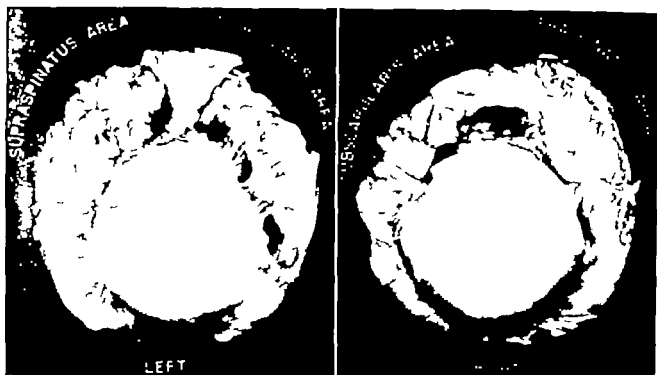


FIG. 128 (Left) J S male white age 59 Left shoulder Profound degenerative changes involving all of the synovials. Note the marked tearing away of the entire musculotendinous cuff from the periphery of the articular margin. Only a few strands of tissue remain intact in the subscapularis area. Observe also the marked hypertrophy of the torn fibers, the deep sulcus, and the widening and thickening of the biceps tendon. On either side of the exit of the tendon the capsular fibers have torn away from the posterior and anterior lips of the intertubercular sulcus so that the exit of the tendon is widened (Right) Right shoulder There are demonstrable advanced degenerative changes of the synovialis in the region of the supraspinatus and infraspinatus regions and to a lesser degree in the subscapularis region of the cuff. Note the recession of the cuff in the supraspinatus area forming a broad sulcus. There is also considerable irregularity and new bone formation in the form of excrescences in the floor of the sulcus. This is an extensive incomplete tear

lower border of the subscapularis tendon. Several ribbonlike strands of tendon fibers in the subscapularis region of the cuff still remained attached to the humeral head. Advanced lesions were also discernible in the biceps tendon particularly the intracapsular portion of the tendon it was frayed thickened and covered with numerous villi. Although the inner and the outer circles of the head of the humerus showed minimal lesions the periphery adjacent to the degenerated cuff was raised and irregular. The sulcus was widened and studded with numerous excrescences of varying sizes.

Not knowing that these specimens were obtained from individuals who were unaware of any disorders of their shoulder

joint from a clinical viewpoint one is apt to interpret these findings as responsible for the painful syndrome so commonly encountered in individuals past middle life. Many workers are of the opinion that partial tears and tendinitis are responsible for symptoms of internal derangements such as torn degenerated menisci produce in the knee joint. That such a syndrome exists in the shoulder joint is common knowledge but in the light of the above observations it is reasonable to conclude that other factors must be present in addition to incomplete tears in the cuff before dysfunction of the scapulohumeral joint is manifested. One is led to believe that pain is not the result of the incomplete tear of the cuff per se but is due

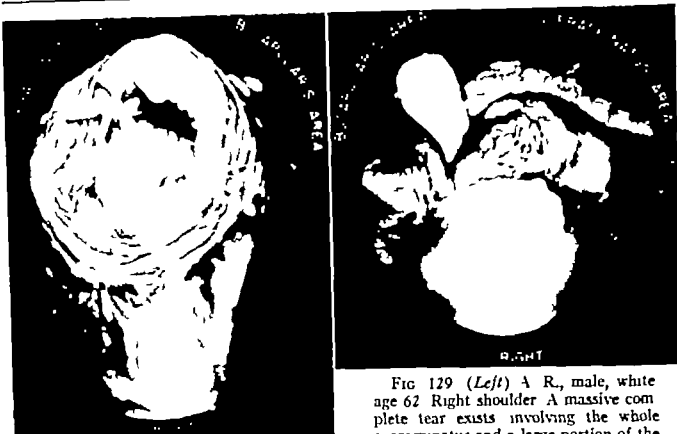


FIG 129 (*Left*) A R., male, white age 62 Right shoulder A massive complete tear exists involving the whole supraspinatus and a large portion of the infraspinatus tendon Note the marked fraying, shredding and degenerative changes in the remaining portion of the cuff (*Right*) The synovial side of the specimen shown at left. Observe the thickness of the biceps tendon which has been completely displaced from the intertubercular groove and lies on a fascial sling formed by the upper border of the intact subscapularis tendon There is also evidence of marked degenerative changes in the synovial membrane of the entire cuff Such an arrangement as is shown by the biceps tendon permits it a free excursion of motion in the new wide tunnel

to impingement of the overlying sensitive subacromial bursa against the coraco-acromial arch during elevation of the arm. Marked thickening and hyperplasia of the torn fibers of the cuff forces the bursal sac against the acromion and the coraco-acromial ligament when the arm is raised above the horizontal position. It is apparent that repeated trauma to the bursa may give rise to the so-called supraspinatus or painful arc syndrome. This explains why excision of the acromion or the bursa or both results in a cure of the symptom complex.

Even more significant than the incomplete tears were the complete tears of the musculotendinous cuff. As previously recorded there were 9 complete tears, ranging in size from 1 centimeter to massive avulsion of the cuff. Six shoulders disclosed only a small portion of the subscapularis tendon and of the infraspinatus and the teres minor tendons to be intact yet these individuals possessed no impairment of shoulder function. Two of these individuals complained of some pain in the right shoulder during motion for approximately six months prior

to death but function was not impaired. Figure 129 shows a right shoulder joint obtained from a male 62 years old a large defect exists in the cuff. The tear includes all of the supraspinatus and a large portion of the infraspinatus tendon. The edges of the tear have retracted medially posteriorly and anteriorly exposing a large segment of the head. In accordance with our present day concept of the extent of a tear in the cuff which is compatible with good function, one is apt to believe that such a tear as this



FIG 130 (Left) M A, female colored age 59 Left shoulder Large complete tear exists, involving all the supraspinatus and infraspinatus portions of the cuff Only the teres minor tendon remains intact. There is also evidence of advanced degenerative changes in the subscapularis region of the musculotendinous cuff. Both tuberosities have been leveled off In this specimen the subacromial bursa has been distended and fixed in order to exhibit marked widening and hypertrophy of its walls. (Right) Right shoulder Note the extensive complete tear involving a portion of the subscapularis tendons and even a portion of the teres minor tendon. In spite of extensive defect in the cuff this patient was able to elevate her arm freely both in the frontal and the coronal planes.

would result in complete loss of abduction power in the shoulder yet the individual had no difficulty in abducting the arm.

More revealing are the two specimens depicted in Figure 130 They were obtained from the shoulders of a female 59 years old The left shoulder (Fig 130 left) discloses an extensive cuff tear extending from the teres minor muscle above to the upper one third of the subscapularis below Also the synovial lining of the remaining cuff exhibits profound degenerative changes Note that both the tuberosities have been leveled off so that the upper end of the humerus forms almost a complete smooth sphere On the other hand the articular cartilage of the head of the humerus shows relatively minimal degenerative changes In Figure 130 left the bursa has been distended and fixed so as to show its thickened walls and its increase in all dimensions

More extensive changes in the musculotendinous cuff were observed in the right shoulder (Fig. 130 right) Only the tendon of the teres minor and approximately half

of the subscapularis tendon remain attached to the head of the humerus As in the left shoulder complete recession of both tuberosities occurred while the articular cartilage of the head of the humerus exhibits minimal degenerative changes Again one would assume that in the face of such extensive pathologic alterations in the stabilizing apparatus of the scapulohumeral joint, dysfunction of the joint must exist. On the



FIG 131 (Left) M W, female, white, age 63 Left shoulder Complete absence of cuff substance in the supraspinatus and infraspinatus areas Only the teres minor tendon and a portion of the subscapularis tendon remain intact Here again we have recession of the tuberosities, so that the upper end of humerus appears like a smooth sphere. (Right) Right shoulder There is again complete destruction of the cuff in the supraspinatus and infraspinatus and part of the subscapularis areas. Only a portion of the teres minor and the subscapularis tendons remain intact. Complete recession of the tuberosities has occurred with eburnation of the exposed bone.

contrary this individual was not aware of any disorder in either shoulder and exhibited good movements in all directions Hence, it becomes obvious that such large defects as noted in this series of cases do not preclude good function

Figure 131 shows both shoulders of a female 63 years of age Again these specimens reveal large extensive tears in the musculotendinous cuff, only a small portion of the subscapularis tendons and the teres minor tendons remain intact Both humeral heads exhibit complete atrophy of the tuberosities and obliteration of the intertubercular sulcus In both instances the degenerated biceps tendon lay on a fascial sling made by the remaining intact portion of the subscapularis tendon fibers Degenerative changes in the articular cartilage of the humeral heads are not severe The subacromial bursa in each instance was large and its walls hyperplastic In each case, the roof of the bursa was adherent to the under surface of the acromion.

BICEPS TENDON

After the fourth decade the biceps tendons manifested degenerative changes which increased in gradient in each successive decade The lesions comprised thickening widening and fraying Lesions of this nature, however did not preclude good painless function of the shoulder joint In Figure 132 the biceps tendon exhibited profound degenerative changes, yet the individual who provided the specimen possessed a normal shoulder from a functional view point As previously stated the severity observed in biceps tendons paralleled those in the musculotendinous cuff (Fig 132) Study of the structures forming the exit of the biceps tendon from the joint cavity



FIG 132 J F male white age 59 Left shoulder Note the advanced degenerative changes in the synovialis and fibers of the musculotendinous cuff and the marked shredding, tearing thickening and widening of the biceps tendon. Observe also that on either side of the exit of the biceps tendon the cuff fibers have torn away from their insertion into the posterior and anterior margins of the intertubercular sulcus so that at no point is the tendon constricted.

revealed that there was tearing away of the cuff and the capsular fibers from the anterior and the posterior walls of the intertubercular sulcus so that the tunnel which the biceps tendon traversed was widened. Such an arrangement afforded ample room for the tendon and permitted free excursion of the head of the humerus on the tendon. In other instances in which the intertubercular sulcus was obliterated by atrophy of the tuberosities the tendon lay in a fascial sling made by the upper border of the tendon of the subscapularis muscle. Here again, the biceps tendon exhibited no evidence of constriction by the surrounding tissues (Fig 129 right).

Figure 133 depicts specimens obtained

from a male aged 73 years. At the time of examination he exhibited no impairment of function in either shoulder. He readily carried his extremities through all arcs of motion. However it was interesting to learn that 12 to 15 years earlier he had considerable pain and stiffness first in the right and then in the left shoulder. After an indefinite period of time there was gradual restoration of function in both shoulders. From the degree of restriction of motion described one was forced to conclude that a frozen shoulder had existed in each extremity. The postmortem specimens disclosed that the intracapsular portions of both the right and the left biceps tendons were absent, while the proximal ends of the extracapsular portions had attained a bony attachment below the lesser tuberosities. Both musculotendinous cuffs exhibited advanced degenerative alterations.

DISCUSSION

Critical assessment of the observations recorded above must be tempered by the knowledge that acute traumatic lesions of the shoulder joint as elsewhere in the body, differ from those acquired over a long period of time resulting from slowly progressing degenerative lesions. In the former group there is sudden disruption of the normal mechanics of the joint with no time for nature to make necessary readjustments of structures remaining intact, so that function of the joint will not be severely impaired. In the latter group as shown in this study such compensating adjustments are possible and take place when slowly progressing degenerative lesions are the causative factors which produce the alterations in the mechanics of the joint. Also it is common knowledge that less extensive tears than those demonstrated in this series, but which were produced suddenly by trauma, may result in marked impairment of shoulder function. One is led to believe therefore that the size of the tear (except in cases of complete avulsion of the cuff) is not the factor which determines the degree



FIG 133 (*Left*) J F male white, age 73 Right shoulder Note that the biceps tendon does not leave the joint through its normal exit. In this instance it has attained a bony attachment to the inferior aspect of the lesser tuberosity. The patient who provided these two specimens gave a history that approximately 12 to 15 years prior to his present and last admission to the hospital he had experienced marked stiffness and pain in both shoulders. The symptom complex described was consistent with that of frozen shoulders. (*Right*) Left shoulder This is a matched specimen of the specimen shown at left. Once more the biceps tendon fails to leave the joint cavity but is attached to the lesser tuberosity. The intra capsular portion of the biceps tendon in both shoulders was absent. Observe the severe in complete tear involving the supraspinatus and infraspinatus areas of the cuff and also the marked hyperplasia of the torn fibers. It is interesting to note that in both illustrations there are only minimal changes in the articular cartilage of each humeral head in spite of the advanced degenerative changes noted in the cuff and the biceps tendon

of impairment of function in the affected shoulder. The writer is of the opinion that loss of function in the scapulohumeral joint is in direct proportion to impairment of muscle balance between the rotator muscles which fix and depress the head in the glenoid cavity and the deltoid muscle. For example a tear implicating all of the supraspinatus and part of the infraspinatus tendon will produce loss of abduction only when the deltoid muscle is sufficiently strong and powerful to overcome the stabilizing action of the torn cuff and pulls the humerus upward under the acromion when abduction is initiated. On the other hand a more extensive tear of the cuff will give rise

to no dysfunction if a relatively less powerful deltoid muscle exists and the remaining rotator apparatus still retains sufficient power to fix and depress the head of the humerus in the glenoid cavity when the deltoid muscle contracts. In other words so long as the intact portions of the musculotendinous cuff (regardless of the size of the tear) retain sufficient power to balance the action of the deltoid muscle no loss of abduction at the scapulohumeral joint ensues.

It becomes obvious that all gradations of dysfunction may exist in different shoulders depending upon the degree of impairment of balance between the rotator apparatus and the deltoid muscle. This provides an

explanation for weak abduction or inability to maintain abduction or total loss of abduction in the glenohumeral joint of middle aged individuals mostly laborers, who possess strong powerful deltoids and at operation exhibit only a small or moderate tear in the cuff. In this group advanced degenerative alterations resulting from physiologic wear and tear aging and occupational traumata weaken the cuff a small or moderate tear in such a cuff may be sufficient to upset completely the muscle balance which permitted normal unrestricted abduction at the glenohumeral joint. Balance having been disturbed varying degrees of impairment of function results. The same size tear as mentioned above or even larger may produce no impairment or less impairment in other individuals usually elderly ones who exhibit relatively less powerful deltoid muscles.

Considering the frequency of extensive tears in the musculotendinous cuff in this series that were compatible with good function one is further led to assume that nature attempts (and in many instances succeeds) to restore impaired muscle balance following traumatic ruptures of the cuff. Therefore it becomes apparent that surgical intervention is justifiable only after sufficient time has elapsed to prove that nature has failed to restore balance.

In the light of the above observations referable to the biceps tendon it is obvious that painless normal movements may be present in shoulder joints with marked degenerative lesions in the biceps tendon and surrounding structures provided that the tendon is given ample room as it passes from an intracapsular to an extracapsular position or if the tendon attains a bony anchorage to the shaft of the humerus thereby

obliterating the tendon tendon sheath gliding mechanism.

SUMMARY AND CONCLUSIONS

1 Degenerative lesions of the articular surface of the head of the humerus are never severe, even in the late decades of life and never equal those observed in the articular cartilage of the glenoid fossa.

2 Extensive, incomplete tears on the synovial side of the musculotendinous cuff are frequently encountered in shoulders with normal function and do not give rise to the painful arc or supraspinatus syndrome in all instances. In this study there were observed 20.8 per cent incomplete subscapular tears and 37.3 incomplete supraspinatus and infraspinatus tears giving rise to no symptoms.

3 Loss of abduction or dysfunction of the scapulohumeral joint does not always occur when large complete tears of the musculotendinous cuff exist. Good function is compatible with massive avulsion of the cuff provided that balance between the deltoid muscle and the remaining intact portion of the rotator cuff is not severely impaired. In this series the incidence of complete tears was 9.3 per cent.

4 From the observations recorded in this investigation it is obvious that nature attempts to restore balance after rupture of the cuff.

5 Surgical intervention is only justifiable after sufficient time has elapsed and nature has failed to restore balance.

6 Advanced degenerative lesions of the biceps tendon are compatible with good function provided that its tunnel of exit from the joint cavity is widened or the tendon attains a bony attachment to the shaft of the humerus.

BIBLIOGRAPHY

- Bankart A S B The pathology and treatment of recurrent dislocations of the shoulder joint Brit J Surg 26 23-29 1938
- Bauer W., and Bennett G A Experimental and pathological studies in the degenerative type of arthritis J Bone & Joint Surg 18 1 18 1936
- Bennett G A and Bauer W A systematic study of the degeneration of articular cartilage in bovine joints Am J Path. 7 399-413 1931
- Benninghoff A. Form und Bau der Gelenkknorpel in ihren Beziehungen zur Funktion I Ztschr f Anat. u. Entwicklungsgesch 76 43-63 1925 II Ztschr f Zellforsch u. mikr Anat., 2 183-362 1925
- Ely L W Inflammation in bones and joints ed. 1 Philadelphia, Lippincott 1923
- Fick R Handbuch der Anatomie des Menschen von Bardeleben Vol. 2 Jena Fischer 1904
- Keyes E L Erosions of the articular surfaces of the knee joint J Bone & Joint Surg 15 369-371 1933
- Nichols E. H and Richardson F L Arthritis deformans J Med Res 21 149-221 1909
- Nicola T Anterior dislocation of the shoulder A new operation J Bone & Joint Surg 11 123 1929
- Parker F Jr Kiefer C C Myers W K., and Irwin R L Histologic changes in the knee joint with advancing age Arch. Path. 17 516-532 1934
- Stevens J H The action of the short rotators on the normal abduction of the arm with a consideration of their action in some cases of subacromial bursitis and allied conditions Am. J. M. Sc 138 870-884, 1909

explanation for weak abduction or inability to maintain abduction or total loss of abduction in the glenohumeral joint of middle aged individuals, mostly laborers, who possess strong, powerful deltoids and at operation exhibit only a small or moderate tear in the cuff. In this group advanced degenerative alterations resulting from physiologic wear and tear aging and occupational traumata weaken the cuff, a small or moderate tear in such a cuff may be sufficient to upset completely the muscle balance which permitted normal unrestricted abduction at the glenohumeral joint. Balance having been disturbed varying degrees of impairment of function results. The same size tear as mentioned above or even larger may produce no impairment or less impairment in other individuals usually elderly ones who exhibit relatively less powerful deltoid muscles.

Considering the frequency of extensive tears in the musculotendinous cuff in this series that were compatible with good function one is further led to assume that nature attempts (and in many instances succeeds) to restore impaired muscle balance following traumatic ruptures of the cuff. Therefore it becomes apparent that surgical intervention is justifiable only after sufficient time has elapsed to prove that nature has failed to restore balance.

In the light of the above observations referable to the biceps tendon it is obvious that painless normal movements may be present in shoulder joints with marked degenerative lesions in the biceps tendon and surrounding structures provided that the tendon is given ample room as it passes from an intracapsular to an extracapsular position, or if the tendon attains a bony anchorage to the shaft of the humerus thereby

obliterating the tendon tendon sheath gliding mechanism.

SUMMARY AND CONCLUSIONS

1 Degenerative lesions of the articular surface of the head of the humerus are never severe even in the late decades of life and never equal those observed in the articular cartilage of the glenoid fossa.

2 Extensive incomplete tears on the synovial side of the musculotendinous cuff are frequently encountered in shoulders with normal function and do not give rise to the painful arc or supraspinatus syndrome in all instances. In this study there were observed 20.8 per cent incomplete subscapular tears and 37.3 incomplete supraspinatus and infraspinatus tears giving rise to no symptoms.

3 Loss of abduction or dysfunction of the scapulohumeral joint does not always occur when large complete tears of the musculotendinous cuff exist. Good function is compatible with massive avulsion of the cuff provided that balance between the deltoid muscle and the remaining intact portion of the rotator cuff is not severely impaired. In this series the incidence of complete tears was 9.3 per cent.

4 From the observations recorded in this investigation it is obvious that nature attempts to restore balance after rupture of the cuff.

5 Surgical intervention is only justifiable after sufficient time has elapsed and nature has failed to restore balance.

6 Advanced degenerative lesions of the biceps tendon are compatible with good function provided that its tunnel of exit from the joint cavity is widened or the tendon attains a bony attachment to the shaft of the humerus.

tendinitis, varying degrees of subacromial bursitis, and lesions of the greater tuberosity, such as sharp or rounded exostoses, associated with or without involvement of the cuff. The 'all inclusive syndrome' is justifiable because the pathogenesis of many of these lesions is closely related. Moreover, the clinical pictures are similar in many respects. Finally, the treatment employed, with few exceptions, is essentially the same.

INCOMPLETE AND COMPLETE TEARS

Clinically tears of the musculotendinous cuff are divided into two categories: (1) partial or incomplete tears and (2) complete tears.

Partial or incomplete tears are lesions which may involve the synovial or bursal surface of the cuff or tendon fibers within the substance of the musculotendinous cuff. Such lesions, however, never establish a direct communication between the joint cavity and the subacromial bursa. In other words, the continuity of the entire thickness of the cuff is never totally interrupted.

Complete tears involve the full thickness of the cuff and establish a direct communication between the joint cavity and the subacromial bursa. They vary in size from barely perceptible lesions to avulsions of the entire cuff.

Clinically the areas of the cuff involved in their order of frequency are: supraspinatus area, supraspinatus and infraspinatus areas, supraspinatus, infraspinatus and teres minor areas, and supraspinatus, infraspinatus, teres minor and subscapularis areas (complete avulsion of the cuff). Tears of the infraspinatus or of teres minor areas singly are encountered rarely, if ever. However, one does occasionally find a lesion involving only the subscapularis area.

These observations differ from those recorded in the study on the synovial side of the cuff. In this investigation lesions in the subscapularis area were demonstrable in 41 per cent of the specimens, and supraspinatus

lesions in 24 per cent. However, tears which involved the entire thickness of the cuff were more frequent in the supraspinatus area. It is apparent therefore that lesions in the subscapularis region of the cuff are less likely to give rise to symptoms than those in the supraspinatus area.

The frequency of the lesions observed in the supraspinatus region of the cuff is dependent upon several factors:

1. **Senescence.** It was noted in the investigation on the musculotendinous cuff that degenerative alterations are grossly demonstrable after the fourth decennium and show an increase in gradient in each subsequent decade. Therefore, senescence plays a major role in the development of these lesions.

Clinically, complete and incomplete tears occur after the fourth decade of life; therefore it is reasonable to assume that these lesions are superimposed upon a degenerated and weakened structure. The highest incidence is found in the sixth age period. Occasionally, they occur in earlier age groups. However, in such instances there is usually a history of violent injury, often resulting in fracture or dislocation of the humeral head.

2. **Attrition.** As pointed out by many observers, the supraspinatus area of the cuff is subjected constantly to frictional trauma as it passes under the acromion and the coraco-acromial ligament when the arm is elevated and abducted and again when the arm is lowered. Continued friction is responsible in part for degenerative alterations in the tendon fibers and initiates a chronic inflammatory process in the floor of the subacromial bursa. Synovial villi tabs and proliferative folds develop in the bursa which impinge against the acromion and the edge of the coraco-acromial ligament as the humeral head passes back and forth under these structures.

3. **Mechanical and Anatomic Factors.** As previously noted, the supraspinatus area, both on the synovial and the bursal sides of

4

Ruptures of the Musculotendinous Cuff

INCOMPLETE AND COMPLETE TEARS

HISTORY OF INJURY

MECHANISM IN PRIMARY TEARS

INCOMPLETE TEARS IN THE SUPRASPINATUS
REGION

COMPLETE TEARS IN THE SUPRASPINATUS
REGION

CLINICAL FEATURES

CONSERVATIVE TREATMENT

SURGICAL TREATMENT

INTRODUCTION

Since the masterful and comprehensive study of Codman many excellent publications have appeared in the literature on lesions of the rotator cuff. Notable contributors on this topic have been Bosworth, Lawrence, Jones, Outland, and Shepherd. Inman, Saunders, and Abbott, McLaughlin, Wilson, Lippmann, and Stevens. Although one may not be in complete agreement with concepts postulated by these observers, they have contributed much valuable information and have provided a keen stimulus for other workers to investigate further this complex region of the shoulder joint.

This revived interest in the subacromial region has culminated in the crystallization of certain clinical entities, such as complete and incomplete ruptures of the supraspinatus tendon, massive and complete avulsion of the rotator cuff, calcareous tendinitis, subacromial bursitis, bicipital tenosynovitis, and frozen shoulder. Because of the intrinsic anatomy of this region, the aforementioned clinical entities are closely interrelated, which has caused considerable confusion in differentiating these lesions. In fact, in many instances a diagnosis can be made only upon visualization of the area. Even then, if one is unfamiliar with the detailed anatomy of the region, one may easily misinterpret the existing pathologic disorder.

In general, there is agreement that degenerative alterations of the musculotendinous cuff are responsible for many lesions in the subacromial region. This is particularly true of ruptures of the cuff, calcareous tendinitis, and frozen shoulder. As noted (see Chap. 2, on Normal Anatomy and Functional Mechanism of the Shoulder Joint and Congenital Abnormalities), the shoulder joint is mechanically inadequate to meet the requirements of a prehensile extremity because of anatomic and phylogenetic factors. During normal joint function, the musculotendinous cuff and the biceps tendon are subjected constantly to harmful stresses and strain whose cumulative effect is responsible for early and progressive degenerative lesions in these structures. For the same reasons, these structures are vulnerable to repeated traumata. In addition to these causative factors, senescence is also responsible to a marked degree for degenerative alterations in the cuff.

RUPTURES (TEARS) OF THE MUSCULOTENDINOUS CUFF

The term supraspinatus syndrome, coined by Bosworth, embodies a group of lesions which directly or indirectly involves the supraspinatus region of the musculotendinous cuff. It includes complete tears of the tendon, incomplete tears, calcareous

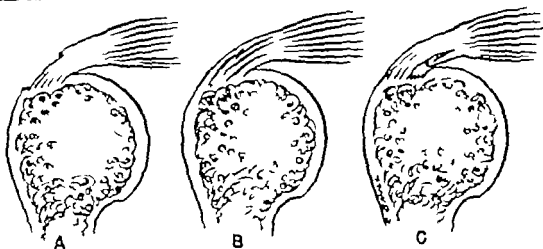


FIG 135 Types of incomplete tears (A) on the bursal side of the cuff, (B) within the substance of the cuff (these lesions are believed by some workers to be the forerunners of calcareous tendinitis) and (C) on the synovial side of the cuff, close to the insertion of the cuff into the head of the humerus

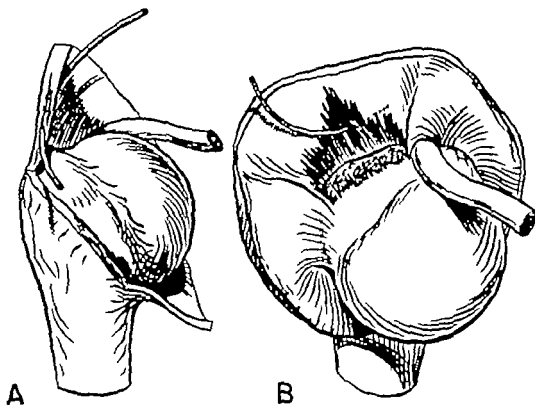


FIG 136 Schematic drawing of actual specimen, showing a large tear on the inner side of the cuff, the fibers have receded from the articular margin and disclose advanced fraying and shredding. The probe is inserted in a longitudinal split in the tendon fibers of the cuff, causing the cuff to laminate into two layers.

MECHANISM RESPONSIBLE FOR PRIMARY TEARS OF THE CUFF

Direct injuries to the point of the shoulder fail to produce tears of the cuff. This region is protected adequately by the over

hanging acromion which receives all impacts directed to this region of the shoulder joint. Tears of the cuff, partial or complete, are caused by indirect forces which subject the musculotendinous cuff particularly in

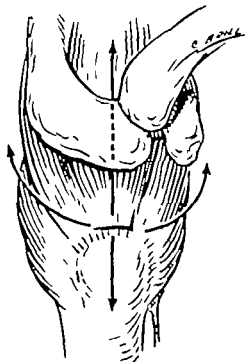


FIG 134 According to Jones rupture of the cuff usually occurs in the supraspinatus tendon which is the weakest portion of the cuff. Divergent forces produced by the rotator muscles cause extension of the tear and prevent healing.

the cuff exhibited more pronounced degenerative changes than other regions. Jones is of the opinion that this impairment of the cuff associated with advancing age is accentuated in intensity by divergent forces which act upon the point where the cuff enters the greater tuberosity. Slight trauma is now capable of producing a cuff tear in the area of greatest impairment generally located in the supraspinatus tendon just proximal to its line of insertion into the facet of the greater tuberosity. Such a defect is subjected to forces pulling in opposite directions, thereby preventing healing and favoring extension of the tear (Fig 134).

The musculotendinous cuff is anatomically and mechanically inadequate to meet the requirements of a prehensile extremity and present-day endeavors which demand the arm to function in an abducted or elevated position. In the discussion on motions

of the shoulder joint it was noted that the prime function of the cuff is to provide a fulcrum for the head of the humerus in the glenoid cavity. This is accomplished by the combined and simultaneous action of the rotator muscles which fix and at the same time depress the humeral head in the glenoid cavity.

In addition the supraspinatus muscle is an active abductor of the arm acting as the short arm of a long lever. In spite of the functional demands made on the supraspinatus muscle, it is the smallest and least powerful of the four component muscles of the cuff.

In the light of the afore mentioned observations one is forced to conclude that the supraspinatus region of the cuff is subjected to great attritional forces resulting in early and severe degenerative alterations in this area. This concept is substantiated by clinical observations and by studies made on cadavers.

4 Occupations. Occupations are recognized predisposing factors in inflicting undue wear and tear on the rotator cuff. Lesions are encountered most frequently in individuals engaged in laborious occupations and athletic endeavors. Men are affected more frequently than women in a ratio of approximately 10 to 1. The right shoulder is more frequently involved than the left.

HISTORY OF INJURY

In all instances a history of injury can be elicited the most frequent being a fall on the outstretched arm. Sometimes the injury is of as mild a nature as picking up some object or elevating the arm suddenly. This is particularly true in the older decades (past the sixth). However severe trauma is an essential prerequisite to initiate the syndrome in individuals in the first three decades of life. Fractures or dislocations of the humeral head are more likely to occur in this age group than a tear of the musculotendinous cuff.

lesions are not encountered frequently. Transverse tearing of the fibers occurs in the cuff slightly proximal to its line of insertion into the tuberosity. When retraction of the torn fibers occurs, it is very slight. Because the edges of the defect are continuous with the adjacent intact cuff, Coleman was of the opinion that most primary tears were transverse in nature, that the edges of the defect were anchored by the adjoining conjoined tendon and that retraction of the muscle made the rent triangular. Also, he believed that the width of the tear was the width of the base and that restoration would be attained if the apex of the triangle were sutured to the middle of the base. McLaughlin, however, is not in agreement with this concept and has offered an entirely different mechanism for the production of triangular and crescentic lesions which are advanced stages of primary transverse tears.

It was of some significance to note that pure transverse complete tears were not disclosed in the investigation on the musculotendinous cuff in cadaver or autopsy specimens. As a rule, small tears were irregular, circular defects in the cuff, some of which exhibited a longitudinal extension of varying length starting from the anterior margin of the defect. Larger tears were either triangular or crescentic. Therefore, it is reasonable to assume that a transverse tear demonstrable at operation is produced by injury (usually in the form of excessive strain) superimposed upon a weakened and degenerated cuff.

2 Pure Vertical Rents or Longitudinal Slits Paralleling the Direction of the Cuff Fibers. Longitudinal tears vary considerably in size. They occur invariably in young individuals who have sustained violent injuries to the shoulder joint. Not infrequently such lesions are associated with fractures or dislocations of the humeral head. In general, the rent runs parallel with the joint cavity through the fibers of the coracohumeral ligament which occupies the interval between the supraspinatus and the

subscapularis muscles and which bridges the intertubercular sulcus.

One case disclosed a tear extending from the greater tuberosity to the base of the coracoid process. As pointed out by McLaughlin, the lesion may continue into the substance of the upper end of the humerus as far as the surgical neck. In these instances the greater tuberosity is fractured at its base and pulled upward and backward by the external rotators while the humeral head is displaced downward and forward by the pull of the subscapularis muscle. It is obvious that the opposing forces of the external rotators and the internal rotator (the subscapularis muscle) tend to separate the edges of a longitudinal rent, thereby preventing healing and favoring extension of the tear.

As was noted in the discussion on transverse tears, complete longitudinal lesions were not observed in cadaver or autopsy specimens. Although vertical separation and shredding of the cuff fibers were common observations, no instance of a complete rent confined to the region of the coracohumeral ligament was encountered. This observation leads one to conclude that such lesions invariably result from violent injury, inflicted upon the strong and healthy musculotendinous cuffs of young individuals capable of resisting transverse tears.

3 Tears with Retraction. According to McLaughlin, excessive strains beyond the capacity of a weakened degenerated cuff produce primary transverse tears.

Repeated and continued strains upon the lesions are responsible for longitudinal splitting of the cuff, starting at one end of the transverse tear and extending proximally parallel with the joint cavity. The longitudinal split usually takes place at the anterior end of the transverse tear. Further traumas, minor or severe, tend to increase the extent of the lesion both in the transverse and the longitudinal axes of the cuff fibers. Also, the longitudinal arm of the lesion is acted upon by the divergent forces of the subscapularis muscle, which pulls the

the supraspinatus region, to great strain beyond its functional capacity. Under normal conditions, strains of this nature would not evoke a tear of the cuff. However, degenerated cuffs are unable to meet such strains and tears of varying degrees result. The sizes and the locations of the tears depend upon the severity of degenerative alterations in the cuff and the amount of strain acting upon it. The usual history obtained is one of falling on the outstretched arm. It is reasonable to assume that the severity of the strain thrown on the supraspinatus area by sudden elevation of the arm is sufficient in itself to cause a tear of the cuff before the individual strikes the ground.

INCOMPLETE TEARS IN THE SUPRASPINATUS REGION OF THE CUFF

These lesions exhibit a diversity of pathologic alterations. Nevertheless they possess a common denominator in that the entire thickness of the cuff is not affected. Essentially they can be divided into four types: (1) tears on the synovial side of the cuff; (2) tears within the substance of the cuff without the involvement of either synovial or bursal surfaces; (3) tears on the bursal side of the cuff; and (4) tears running parallel with the cuff fibers (Fig. 135).

1 **Tears on the Synovial Side of the Cuff (Rim Rents).** Such lesions involve the deep tendon fibers which, together with the synovialis, are torn from their bony insertion (Fig. 136). Retraction of the fibers gives rise to a mobile tab of tissue which, as emphasized by McLaughlin and Bosworth, may produce symptoms of internal derangement of the shoulder joint comparable with internal derangement of the knee associated with lesions of the menisci.

In acute and subacute lesions an active reparative process may be initiated which produces pronounced thickening of the torn fibers at the base of the tear. Such a thickened and hypertrophied portion of the cuff is responsible for the soft crepitus and jog often demonstrable in affected shoulder

joints. The soft crepitus and jog are discernible when the thickened portion of the cuff impinges upon and then passes under the acromion or the falciform edge of the coraco-acromial ligament. Chronic lesions also may disclose some thickening of the torn fibers and base of the tear resulting from advanced fibrosis caused by the increased friction in this region. Occasionally there may be considerable thinning in the impaired portion of the cuff.

2 **Tears Within the Substance of the Cuff Without Involvement of the Synovial or Bursal Surfaces.** Tears may occur in the central fibers, causing the cuff to laminate into two or more layers (Figs. 135 and 136).

3 **Tears on the Bursal Side of the Cuff.** Such lesions reveal tearing of the superficial fibers just proximal to their point of insertion into the facet of the greater tuberosity (Fig. 135A). Fresh lesions always demonstrate the distal remnants of the cuff fibers still attached to the tuberosity. The floor of the subacromial bursa is involved in all these lesions. The constant irritation produced by the defect in the cuff frequently causes pronounced thickening of the bursal floor. Numerous villi and thick synovial folds covering the bursal floor are frequent findings. It is obvious that such lesions hinder smooth joint motion and give rise to symptoms similar to those found in tears on the synovial side of the cuff.

4 **Tears Running Parallel with the Cuff Fibers.** These lesions are the least frequently encountered. Here again the overlying bursa always is affected and discloses changes similar to those noted above.

COMPLETE TEARS IN THE SUPRASPINATUS REGION OF THE CUFF

McLaughlin has given us the most comprehensive classification of these lesions. He divides them into four groups: pure transverse ruptures, pure vertical rents, tears with retraction, and massive avulsion of the cuff.

1 **Pure Transverse Ruptures.** These

lesions are not encountered frequently. Transverse tearing of the fibers occurs in the cuff slightly proximal to its line of insertion into the tuberosity. When retraction of the torn fibers occurs, it is very slight because the edges of the defect are continuous with the adjacent intact cuff. Coolman was of the opinion that most primary tears were transverse in nature, that the edges of the defect were anchored by the adjoining conjoined tendon and that retraction of the muscle made the rent triangular. Also, he believed that the width of the tear was the width of the base and that restoration would be attained if the apex of the triangle were sutured to the middle of the base. McLaughlin, however, is not in agreement with this concept and has offered an entirely different mechanism for the production of triangular and crescentic lesions which are advanced stages of primary transverse tears.

It was of some significance to note that pure transverse complete tears were not disclosed in the investigation on the musculotendinous cuff in cadaver or autopsy specimens. As a rule small tears were irregular, circular defects in the cuff some of which exhibited a longitudinal extension of varying length, starting from the anterior margin of the defect. Larger tears were either triangular or crescentic. Therefore it is reasonable to assume that a transverse tear demonstrable at operation is produced by injury (usually in the form of excessive strain) superimposed upon a weakened and degenerated cuff.

2. Pure Vertical Rents or Longitudinal Slits Paralleling the Direction of the Cuff Fibers. Longitudinal tears vary considerably in size. They occur invariably in young individuals who have sustained violent injuries to the shoulder joint. Not infrequently such lesions are associated with fractures or dislocations of the humeral head. In general the rent runs parallel with the joint cavity through the fibers of the coracohumeral ligament which occupies the interval between the supraspinatus and the

subscapularis muscles and which bridges the intertubercular sulcus.

One case disclosed a tear extending from the greater tuberosity to the base of the coracoid process. As pointed out by McLaughlin, the lesion may continue into the substance of the upper end of the humerus as far as the surgical neck. In these instances the greater tuberosity is fractured at its base and pulled upward and backward by the external rotators while the humeral head is displaced downward and forward by the pull of the subscapularis muscle. It is obvious that the opposing forces of the external rotators and the internal rotator (the subscapularis muscle) tend to separate the edges of a longitudinal rent, thereby preventing healing and favoring extension of the tear.

As was noted in the discussion on transverse tears, complete longitudinal lesions were not observed in cadaver or autopsy specimens. Although vertical separation and shredding of the cuff fibers were common observations, no instance of a complete rent confined to the region of the coracohumeral ligament was encountered. This observation leads one to conclude that such lesions invariably result from violent injury, inflicted upon the strong and healthy musculotendinous cuffs of young individuals capable of resisting transverse tears.

3. Tears with Retraction. According to McLaughlin, excessive strains beyond the capacity of a weakened degenerated cuff produce primary transverse tears.

Repeated and continued strains upon the lesions are responsible for longitudinal splitting of the cuff, starting at one end of the transverse tear and extending proximally parallel with the joint cavity. The longitudinal split usually takes place at the anterior end of the transverse tear. Further traumas, minor or severe, tend to increase the extent of the lesion both in the transverse and the longitudinal axes of the cuff fibers. Also, the longitudinal arm of the lesion is acted upon by the divergent forces of the subscapularis muscle which pulls the

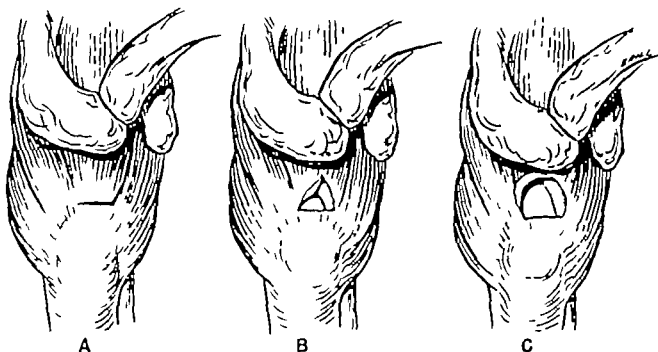


FIG. 137 McLaughlin's concept of the formation of cuff defects. (A) First a transverse tear or rupture of varying size occurs as the result of trauma inflicted on a degenerated cuff. (B) Repeated traumata cause extension of the tear both in the transverse and longitudinal axis. (C) Divergent forces act on the longitudinal arm of the tear: the subscapularis pulls the anterior edge of the tear forward and the external rotators pull the posterior edge backward producing a triangular or crescentic effect.

anterior margin of the tear forward and the external rotators which pull the posterior margin backward thereby producing the typical triangular or crescentic defect disclosed at operation (Fig. 137).

It is apparent from the afore mentioned observations that a triangular or crescentic lesion is a combination of a transverse and longitudinal tear. In old lesions a smooth hypertrophied margin is discernible around the entire defect on the synovial side of the cuff. This is nature's frustrated attempt to limit the defect and is really a reparative process in the torn synovialis proximal to the defect in the cuff fibers. Codman designated this thickened synovial margin the "falciiform ligament." As a result of this attempt at repair the retracted edges of the gap are smooth. A distinct transverse or longitudinal rent is no longer demonstrable so that one may readily misinterpret the lesion as a transverse tear superimposed by retraction of the muscle (the supraspinatus muscle). As previously stated this concept differs from that of Codman who believed

the above defects to be the result of retraction of the supraspinatus muscle following a transverse tear in its tendinous insertion.

4 Massive Avulsion of the Cuff Such lesions comprise avulsion of a large segment of the cuff (usually all the external rotator tendons are involved) with retraction. The longitudinal tear takes place in the interval between the supraspinatus and the subscapularis muscles through the fibers of the coracohumeral ligament. Occasionally the lesion includes part of the subscapularis tendon. In these instances the longitudinal tear extends medially through the subscapularis muscle. Not infrequently the torn and retracted cuff hangs like a curtain between the head of the humerus and the glenoid cavity. McLaughlin applied the term complete avulsion to those cases in which the entire musculotendinous cuff (all four rotator tendons) is avulsed and retracted from the humerus.

Lesions as described above occur in the aged as a rule although occasionally they are noted in younger individuals who sus-

tained violent trauma. They are often concomitant lesions of anterior dislocations and often responsible for chronic subluxation of the shoulder.

CLINICAL FEATURES

1 Complete Tear in the Supraspinatus Region of the Cuff Codman's classic description of the clinical features of a rupture of the supraspinatus tendon is so complete and comprehensive that no one as yet has been able to add anything new. One is forced to draw heavily from this rich source of information when considering the diagnostic features of this clinical entity.

At this point for the sake of facilitating comprehension of the clinical picture that is to follow some repetition referable to the anatomy of the musculotendinous cuff is justifiable. All four rotator tendons are welded together with the fibrous capsule into one musculotendinous sheath which inserts into the upper half of the anatomic neck of the humerus. The fibers of the four tendons interlace so intimately with one another and with the fibers of the capsule that it is impossible to separate the components of any one tendon at its point of insertion even with sharp dissection.

Since the supraspinatus region of the cuff is subjected to the greatest amount of strain generally all complete tears involve this area. At first the rupture may involve only the supraspinatus region of the cuff, then as the result of further strain it may extend laterally to involve the infraspinatus and the teres minor regions. Medial extension of the tear will involve the subscapularis area. On the other hand massive or complete avulsion of the cuff may occur at the time of the initial trauma. It becomes obvious that the clinical features are dependent upon time, the severity of the lesion and the extent of secondary pathologic alterations that ensue in the subacromial bursa, the humeral head, the tuberosities and the biceps tendon.

The pertinent clinical features of complete tears are (1) age, (2) occupation

(3) a history of injury, (4) pain, (5) impaired function and (6) the local clinical features.

AGE Such lesions usually occur past middle age, during which period there is indisputable evidence of attritional changes in the musculotendinous cuff. When complete tears occur in younger individuals there is always a history of violent trauma.

OCCUPATION In general, the lesions occur in individuals who perform strenuous work. These individuals not only inflict constantly minor trauma to their cuffs, thereby favoring progressive degenerative disorders, but also by the very nature of their work are vulnerable to trauma which may precipitate a complete tear.

HISTORY OF INJURY A history of injury is invariably available. Falling on the outstretched arm is the most common form of injury. Next in frequency is one of sudden abduction of the extremity while grasping a heavy object" (Codman). Occasionally it is difficult to correlate the clinical picture with the mechanism of production which may appear minor and insignificant. However, if one visualizes the extensive degenerative abnormalities present in the cuff of the person affected, it becomes obvious that minor trauma is sufficient to produce extensive avulsion of such a damaged structure. The writer has seen in elderly individuals two cases of massive avulsion of the cuff resulting from shock therapy.

PAIN Sudden sharp pain at the time of the injury is a constant and characteristic feature. Although the pain is localized at the tip of the shoulder, it is also referred to the insertion of the deltoid muscle. Often the patient will volunteer that a sensation of something 'snapping' or 'tearing' was felt in the shoulder. Within a few hours the acute pain subsides only to recur with greater intensity in the next 6 or 12 hours. For the next several days (4 to 7 days) there is an increase in the intensity of the pain which then gradually diminishes.

Hemorrhage continues to distend these structures for several days thereby produc-

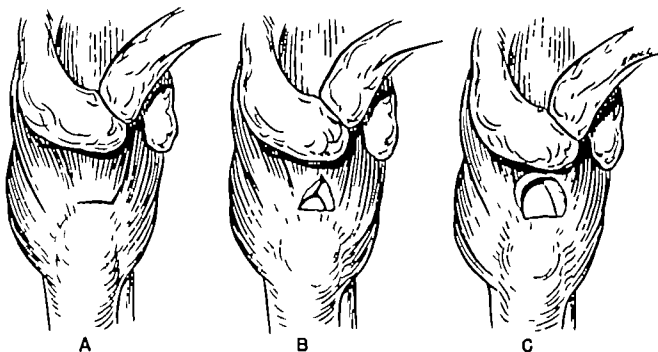


FIG 137 McLaughlin's concept of the formation of cuff defects. (A) First a transverse tear or rupture of varying size occurs as the result of trauma inflicted on a degenerated cuff (B) Repeated traumata cause extension of the tear both in the transverse and longitudinal axis (C) Divergent forces act on the longitudinal arm of the tear the subscapularis pulls the anterior edge of the tear forward and the external rotators pull the posterior edge backward producing a triangular or crescentic effect.

anterior margin of the tear forward and the external rotators which pull the posterior margin backward thereby producing the typical triangular or crescentic defect disclosed at operation (Fig 137)

It is apparent from the afore-mentioned observations that a triangular or crescentic lesion is a combination of a transverse and longitudinal tear. In old lesions a smooth hypertrophied margin is discernible around the entire defect on the synovial side of the cuff. This is nature's frustrated attempt to limit the defect and is really a reparative process in the torn synovialis proximal to the defect in the cuff fibers. Codman designated this thickened synovial margin the falciform ligament. As a result of this attempt at repair the retracted edges of the gap are smooth. A distinct transverse or longitudinal rent is no longer demonstrable so that one may readily misinterpret the lesion as a transverse tear superimposed by retraction of the muscle (the supraspinatus muscle). As previously stated this concept differs from that of Codman who believed

the above defects to be the result of retraction of the supraspinatus muscle following a transverse tear in its tendinous insertion.

4 Massive Avulsion of the Cuff Such lesions comprise avulsion of a large segment of the cuff (usually all the external rotator tendons are involved) with retraction. The longitudinal tear takes place in the interval between the supraspinatus and the subscapularis muscles through the fibers of the coracohumeral ligament. Occasionally the lesion includes part of the subscapularis tendon. In these instances the longitudinal tear extends medially through the subscapularis muscle. Not infrequently the torn and retracted cuff hangs like a curtain between the head of the humerus and the glenoid cavity. McLaughlin applied the term complete avulsion to those cases in which the entire musculotendinous cuff (all four rotator tendons) is avulsed and retracted from the humerus.

Lesions as described above occur in the aged as a rule although occasionally they are noted in younger individuals who sus-

role in abduction of the arm. They establish a fulcrum for the humeral head in the glenoid cavity by holding the head firmly against the glenoid and, at the same time, depressing it. They guide or direct the pull of the deltoid muscle. In addition, the supraspinatus is also an abductor of the arm but does not initiate the abduction. It acts simultaneously with the deltoid throughout the entire arc of abduction. Such a mechanism is essential in order to attain full and powerful abduction and elevation of the arm. If there exists a massive tear of the cuff and if the rotator apparatus which remains intact is not sufficient to stabilize the humeral head, the humeral head is unable to obtain a fulcrum on the glenoid cavity, being displaced under the acromion by powerful contractions by the deltoid in a vertical direction. No scapulohumeral motion is now possible, although the scapula can rotate on the thorax from 60° to 70° . Cuffs with lesser tears may provide a fulcrum for the humerus sufficient to permit weak abduction in the scapulohumeral joint. The fulcrum, however, is not sufficiently stable to allow abducting the arm against resistance or to maintain abduction. Small tears which do not severely lessen the efficiency of the cuff are compatible with smooth powerful abduction particularly if the impairment does not give rise to symptoms of internal joint derangement.

It was noted in the investigation dealing with degenerative lesions (p. 105) that the size of the cuff tear (except in cases of complete avulsion of the cuff) is not the factor which determines the extent of dysfunction in the involved extremity. Loss of function (primarily that of controlled powerful elevation of the arm) of the scapulohumeral joint is in direct proportion to the impairment of muscle balance between the rotator muscles which fix and depress the head of the humerus in the glenoid fossa and the deltoid muscle. This is also true of the power to abduct the arm against resistance and to maintain abduction.

This affords an explanation for the marked impairment of function in individuals with strong powerful deltoid muscles who suffer only small or moderate tears of the cuff, and for good function in elderly individuals with extensive or massive avulsions of the cuff who possess small and relatively less powerful deltoid muscles. In the first group, which mostly includes laborers past the fourth decade, the deltoid muscle is unusually well developed, strong and powerful, while the rotator apparatus may show extensive degenerative lesions resulting from physiologic wear and tear and repeated occupational traumata. A small or moderate tear (for example, one involving the supraspinatus and a portion of the infraspinatus muscle) in this group may disturb sufficiently the balance between the deltoid and the rotator muscles so that pronounced impairment of function results as total inability to abduct the arm or weak abduction and inability to maintain abduction.

On the other hand, extensive tears of the cuff in individuals with less powerful deltoid muscles may give rise to little or no apparent dysfunction, provided that power in the remaining intact rotator cuff is sufficient to stabilize the head of the humerus by balancing the contraction of the deltoid muscle.

Faulty scapulohumeral rhythm is a constant feature of complete tears of the cuff. It also may be exhibited with impairment of function resulting from loss of balance between the deltoid and the rotator muscles, by incomplete tears and calcareous tendinitis, if pain and muscle spasm exist. Normally, during elevation and abduction motion is smooth, simultaneous and continuous in both the scapulothoracic and the scapulohumeral joints. There is a constant relationship of scapular to humeral motion, the ratio being two of humeral motion to one of scapular motion (Inman, Saunders and Abbott). In persons with cuff tears which impair function this relationship is altered. As the arm is raised to the hori-

ing increasing 'tension pain' With cessation of bleeding the hemorrhage is absorbed slowly, and the pain subsides During this acute painful period there is marked protective muscle spasm which precludes adequate examination of the part In the latter stage the pain is constant but less intense It is projected to the insertion of the deltoid muscle and at times to the anterior aspect of the arm It is accentuated by activity and interferes with the individual's sleep

IMPAIRED FUNCTION may result from abnormal mechanical factors or pain which produces muscle spasm on attempts at motion In the acute stages pain and muscle spasm may be so pronounced that the severity of the lesion cannot be ascertained by physical examination This may be true in extensive lesions of the cuff or in such trivial ones as tears of a few of its fibers in the supraspinatus and the infraspinatus regions

Severe pain occurs in instances of minor cuff damage only when the torn portion of the cuff passes the acromion or the coraco-humeral ligament during abduction of the arm Immediate relief takes place after the tender tissues have passed beneath these structures This painful arc of movement is between 80° and 110° or 120° As the arm is lowered to the side pain is again felt at 120° and disappears at 80° If the rupture is not too extensive free painless abduction is possible if the pain factor is eliminated This can be accomplished readily by infiltration of the torn cuff with procaine (5 cc of a 1 per cent solution)

Also depression of the humeral head manually by the examiner as the arm is abducted will prevent impingement of the impaired cuff against the coraco-acromial arch hence no pain is felt Free painless motion without restriction in these instances can be demonstrated by having the patient stoop to a horizontal position with the knees extended and the arms hanging loosely toward the floor In this position all muscles around the shoulder girdle, including the deltoid are relaxed. Thus the patient re-

quires little effort to swing the arm into a position of complete elevation If the examiner supports the weight of the arm as the patient regains the upright position complete elevation can be maintained by the patient, even if the supraspinatus is torn.

The degree to which normal function is impaired depends upon the extent of the cuff tear and the associated secondary changes in the adjacent structures. Codman expressed the belief that all complete supraspinatus tears should be repaired, and the repair should be done immediately The writer, together with other observers, is not in agreement with this concept Complete tears of the cuff within certain limits are compatible with normal and painless shoulder motion In the investigation on shoulder joints, obtained postmortem from individuals who were not aware of any impairment of function prior to their death, it was noted that nine shoulder joints disclosed complete tears of the cuff ranging in size from 1 cm. to massive avulsion

Moreover the writer has explored shoulder joints which exhibited clinically the cardinal features of a bicipital tenosynovitis. In addition to this lesion complete tears of the supraspinatus region of the musculotendinous cuff were demonstrable The tears ranged in sizes from 1 to 4 cm For investigative reasons the cuff tears were not repaired The biceps tendon was transplanted to the coracoid process In all these cases there was complete restoration of free painless joint motion

As previously noted in the acute stages it is impossible to make a correct diagnosis of complete rupture of the cuff because of the associated pain and muscle spasm. In the later stages if the tear is extensive enough to prevent firm fixation of the humeral head in the glenoid cavity impaired function is discernible This is manifest by an inability to abduct or maintain abduction and faulty scapulohumeral rhythm

Through the musculotendinous cuff the rotator muscles normally play an important

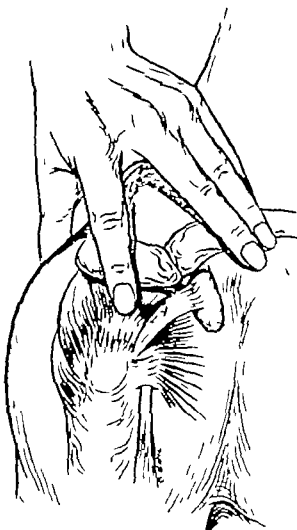


FIG 140 The tip of the forefinger in position to elicit point of maximum tenderness in cases with rupture of the supraspinatus region of the cuff. The finger drops into the sulcus. Pressure over this region causes severe pain. The eminence may be palpated just external to the sulcus.

of the tuberosity. Tenderness over the mid point of the outer surface of the humeral head is indicative of involvement of the teres minor region of the cuff. Also tenderness can always be elicited at the insertion of the deltoid. In fact many of these patients will volunteer to say that there is pain in this region but fail to mention that there is a tender area at the point of the shoulder. In the acute stage there is marked sensitivity at the site of the tear. Sensitivity diminishes with time and in long standing cases it is not a very significant sign (Figs 138-141).



FIG 141 Roentgenograph of patient with large tear of the cuff. Note the prominent irregular tip of the greater tuberosity and slight condensation of cortical bone just medial to the eminence.

Jog and Soft Crepitus A characteristic catch or jog and soft crepitus can be felt by the patient and the examiner as the arm is abducted beyond the horizontal position. They are produced when the impaired portion of the cuff and the tuberosity (eminence) impinge upon and then pass under the acromion or the coraco-acromial ligament. Both jog and crepitus are again palpable when the arm is lowered as the torn and sensitive cuff and the tuberosity pass from under the coraco-acromial arch. Although these signs (together with the afore-mentioned clinical features) are presumptive evidence of a complete tear of the cuff they may be demonstrable also in other lesions responsible for internal derangement of the shoulder joint.

Atrophy of the Rotator Muscles Atrophy of the pinnatii is a constant sequel of this

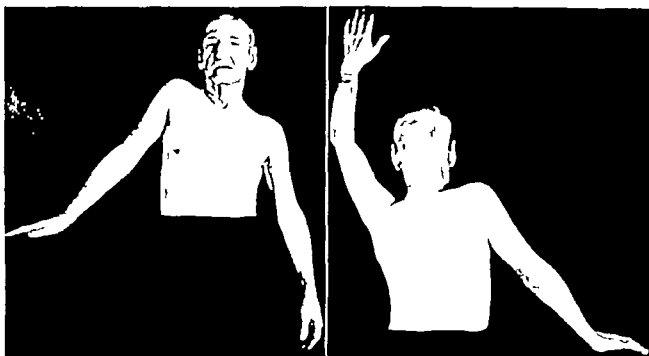


FIG 138 Patient showing maximum range of abduction that was possible with large complete rupture of the cuff, implicating all the supraspinatus and one-third of the infraspinatus regions of the cuff



FIG 139 Hands in position for examination of shoulder. The thumb lies just below the spine of the scapula while the tip of the forefinger is just anterior to the acromion the other three fingers hold the clavicle. The entire shoulder girdle now is firmly grasped by the hand and any motion in the glenohumeral joint is readily discernible.

zonal position one notes that all motion takes place in the scapulothoracic joint while the glenohumeral joint is fixed in a position of flexion. From the horizontal position elevation of the arm is completed by motion entirely through the glenohumeral joint. When the arm is lowered the relationship between the capula and the humerus is not altered until the horizontal position is reached. In other words the arm approaches the horizontal from the position of complete elevation with the glenohumeral joint fixed in the position of extension; all motion taking place in the scapulothoracic joint. From the horizontal the arm is suddenly dropped to the side; this last phase of motion taking place in the glenohumeral joint.

LOCAL CLINICAL FEATURES

Tenderness. Marked tenderness can invariably be elicited over the tip of the greater tuberosity (superior facet). When the lesion extends into the infraspinatus region point tenderness can be demonstrated slightly below the lateral to the tip



FIG 145 (Left) Supraspinatus region of cuff at the point of insertion into the head of the humerus in a male 56 years old, with no shoulder dysfunction. Observe the advanced shredding, tearing fibrillation of the tendon fibers increase in connective tissue formation and the appearance of numerous blood vessels. ($\times 62$)

FIG 146 (Right) Cuff disclosing hyalinization of the degenerated fibers and profound increase in connective tissue elements. Case of male 60 years old with no shoulder dysfunction ($\times 62$)

entity. The degree of atrophy is dependent upon the severity and the duration of the lesion. When the affected shoulder joint is compared with the opposite normal side one will note that the muscles exhibiting most pronounced atrophy are the supraspinatus, the infraspinatus, the teres minor and the trapezius muscles. The deltoid muscle may disclose apparent or real hypertrophy. From what was noted previously in the discussion on impaired function it is apparent that when the cuff fails to provide an adequate fulcrum, the deltoid undergoes powerful contractions with each attempt to abduct or elevate the extremity. Such excessive demands result in hypertrophy of the deltoid muscle.

Radiographic Examination Unless there is involvement of the osseous elements of the humerus radiographic examination reveals no significant observations. In the presence of marked muscle spasm in the early stages the humeral head may be found high in the glenoid cavity.

Long-standing cases may show some increased localized density in the tip of the greater tuberosity and at times some spurring in this region. In such instances definite extoses also may be demonstrable on the tip of the greater tuberosity (Fig 141).

Cases with massive and complete long-standing avulsions of the cuff may reveal "recession" of the tuberosities and hypertrophic changes on the outer surface of the humeral shaft. Anteroposterior, lateral and oblique views may be necessary to show the above lesions (Fig 142).

Presence of Fluid Increased amounts of synovial fluid can be demonstrated in most instances of old complete tears. It is only demonstrable when the arm is elevated as far as is possible and is palpable in the subacromial bursa beneath the deltoid muscle. When the arm is at the side the fluid gravitates to the inferior regions of the capsule. Continued distention of the bursa in old cases produces marked thickening of the bursal walls. This was a common observa-



FIG 142 Roentgenograph of the head of the right humerus of a patient with old complete tear of cuff (postmortem specimen) Observe the recession and atrophy of the greater tuberosity and irregularity of the superior and lateral surface of the shaft of the humerus resulting from the formation of bony excrescences.

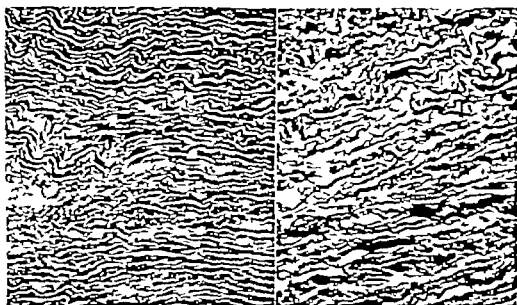


FIG 143 (Left) Supraspinatus tendon showing no degenerative alterations of an individual 30 years old Observe the continuous wavy pattern of the collagenous fibers and the paucity of cellular elements and blood vessels. ($\times 75$)

FIG 144 (Right) Supraspinatus region in an individual 42 years old Note moderate degenerative changes manifested by loss of the wavy pattern of collagenous fibers fibrillation and shredding of fibers note also the increase in cellular elements blood vessels and connective tissue. ($\times 150$)

subside, the nature of the syndrome varies with the severity of the lesion

It has been demonstrated conclusively that not all complete tears give rise to symptoms, nor do they always impair function. Lesions extensive enough to impair function by failure of the rotator apparatus to balance the action of the deltoid muscle are recognized readily because all the previously mentioned cardinal features of this syndrome are usually present

2. **Incomplete Tears in the Supraspinatus Region of the Cuff** The pertinent clinical features of this entity are similar to those of complete tears of the cuff. As a rule, they are less pronounced, and such local signs as the presence of an eminence and a sulcus may be absent in some instances. Inasmuch as there is no communication between the joint cavity and the subacromial bursa, it is impossible for synovial fluid to accumulate under the deltoid muscle. Hence it is not demonstrable.

At times, accurate diagnosis of an incomplete tear of the cuff may be very difficult. However, there is one cardinal feature which distinguishes it from complete tears. Good power of abduction and elevation is always present in incomplete tears. Poor and weak power of abduction and elevation is a characteristic feature of complete tears sufficiently large to cause impaired function. However, even at the risk of criticism for repetition, it must be restated that a multitude of entities present clinical pictures comparable with that of incomplete tears of the cuff, making visualization of the area imperative in order to arrive at a correct diagnosis.

TREATMENT

CONSERVATIVE TREATMENT OF INCOMPLETE TEARS

1. **Recent Lesions.** During the acute stage treatment should be entirely palliative, directed toward alleviation of pain, overcoming muscle spasm, restoration of motion, prevention of muscular atrophy and

prevention of disturbing sequelae. Sedatives may have to be administered (especially before retiring and for several days) until the acute pain subsides. Infiltration of the supraspinatus region of the cuff with 5 cc. of 1 per cent procaine solution often gives dramatic relief from pain, relieves muscle spasm and restores motion. It may be necessary to repeat the procedure three or four times on successive days.

Rest of the part is essential, particularly in severe lesions. This is best attained by placing the arm in a sling. However, one should guard against complete immobilization of the arm, because serious sequelae may occur. At frequent and regular intervals during the day (every 2 or 3 hours) the arm should be removed from the sling and the patient should be encouraged to put the arm through a routine of gravity free pendulum exercises for short periods of time (3 to 5 minutes). Physical therapy in the form of radiant heat and gentle massage may be added to the program. With cessation of the acute symptoms (after 4 or 5 days) the sling is discarded, and wall crawling exercises are started. Caution must be taken not to allow the patient to force the arm beyond the tolerance of pain and fatigue.

Mobility is a very essential part of the program, for one must remember that these lesions occur in individuals past middle life in whom degenerative cuff changes are present in varying severity. Immobilization together with degenerative alterations in the cuff may initiate inflammatory processes which terminate in frozen shoulders.

At no stage during the treatment is any type of passive manipulation of the shoulder justifiable. Such procedures only inflict further damage to the cuff and favor further formation of adhesions.

Resumption of normal arm motions must be encouraged as soon as the acute symptoms subside. Progressive use up to the point of tolerance and fatigue is the secret of successful treatment.

If such a program is adhered to strictly

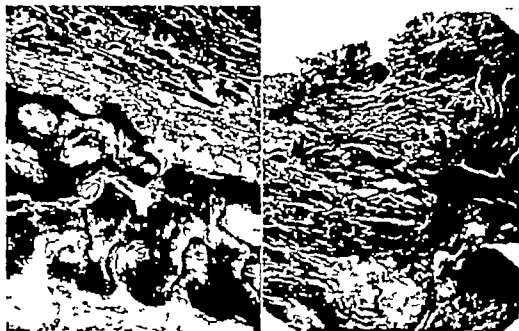


FIG 147 (Left) Supraspinatus region of cuff in a female 56 years old. Note bone formation within the substance of the cuff. The individual was not aware of any shoulder impairment. ($\times 62$)

FIG 148 (Right) Portion of a ruptured supraspinatus tendon of a female aged 48. Note the extensive degenerative alterations of the cuff fibers. Although more severe, the nature of the abnormalities is similar to that depicted in Figure 144. ($\times 62$)



FIG 149 Cuff shown in Figure 148 ($\times 152$)

tion in cadaver and autopsy specimens (Fig 130, left)

Eminence and Sulcus Tears extensive enough to give rise to impaired function can often be diagnosed by the presence of an *eminence* and a *sulcus*.¹ The *sulcus* is a palpable defect at the site of rupture in the musculotendinous cuff. The *eminence* is the normal prominence of the tuberosity, to which may still be attached some remnants of the insertion of the cuff. It is located and can be readily palpated just anterior to the edge of the acromion when the arm is in dorsal flexion. The jog and soft crepitus is caused by the passing of the eminence and the *sulcus* under the acromion and the coraco-acromial ligament.

In summary one is safe in saying that in the early acute stage when severe pain and muscle spasm obscure the clinical picture it is not possible to make a positive diagnosis of complete tear in the supraspinatus region of the cuff. After the acute symptoms

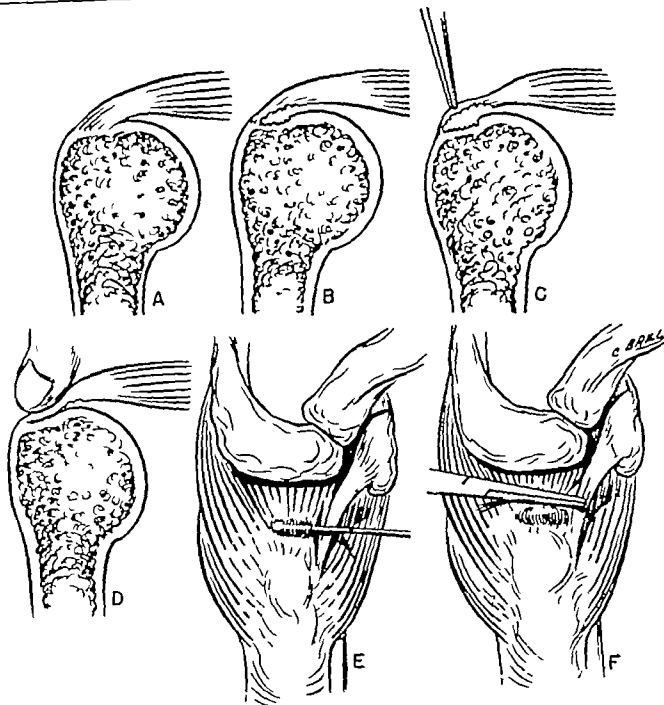


FIG. 150 Aids in arriving at a diagnosis of incomplete tears of the cuff. (A) Normal insertion of the cuff into the humeral head. fibers are firmly attached and cannot be lifted off. (B) Rim rent or incomplete tear. (C) Outer fibers of cuff in incomplete tears can be lifted from the head of the humerus. (D) Finger can palpate sulcus and irregularities on the humeral head through the thinned cuff. (E) Longitudinal split in cuff will permit probe to be inserted into the side of the cuff, also permitting visualization of the inner surface of the cuff. (F) Blister is raised when instrument is passed laterally over the cuff (Redrawn with modifications from McLaughlin)

1 Normally upon abduction of the arm in various planes of rotation the musculotendinous cuff passes under the coracoacromial arch without a wrinkle on its superficial surface. However if a rim rent

is present, as the arm is abducted a wrinkle or "blister" forms at the site of the lesion as it approaches the falciform edge of the coracoacromial ligament or edge of the acromion

the great majority of the patients will attain complete, painless restoration of function within from three to six weeks. Operative treatment is warranted if satisfactory improvement is not attained but it is never justified before ten to twelve weeks.

2 Old Lesions. Old incomplete tears deserve a fair trial at conservative measures before surgical intervention is contemplated. Many cases respond favorably under a carefully planned regimen of treatment. It is of prime importance to gain the confidence of the patient in the execution of such a program. Objectives to be achieved are restoration of complete motion and alleviation of pain.

Relief from pain is attained by such measures as radiant heat, gentle massage and infiltration of the area of maximum tenderness with 1 per cent procaine. Rest is another essential feature of the program. Painful arcs of motion should be avoided and restraint from strenuous activity should be insisted upon.

Restoration of motion can be achieved by graduated pendulum and wall-crawling exercises performed within the tolerance of pain and fatigue. At no time should one resort to manipulative procedures in an attempt to restore full range of motion. Formation of more adhesions following manipulation invariably results in greater restriction of motion.

With such a program satisfactory results should be attained in from four to six weeks although in some instances several months of intensive therapy may be necessary before satisfactory recovery is obtained. Failure to attain improvement is sufficient justification for operative intervention particularly if pain, muscle spasm and restriction of motion are of such severity that joint function is markedly impaired.

CONSERVATIVE TREATMENT OF COMPLETE TEARS

During the acute stage treatment is similar to that described for incomplete tears. As previously noted many minor

complete tears give no functional impairment after the acute painful symptoms have subsided. More severe tears may improve spontaneously without too much residual disability, a matter acceptable to both the patient and the surgeon.

These clinical observations make it mandatory that a sufficient interim be allowed between initial injury and decision for surgical intervention. From six to eight weeks is not too long an interval to elapse before operative repair of the lesion is undertaken. McLaughlin has recorded that his end result studies of early and late repairs revealed no appreciable differences in functional recovery. This concept is not in agreement with Codman, Mosely and Bosworth who are of the opinion that the results of early operation are superior to those of late operation. The author's end results substantiate McLaughlin's observations.

Other factors should be weighed thoroughly before one decides upon surgical intervention. Elderly inactive people with marked functional impairment of the shoulder who tolerate the disability without too much discomfort should not be subjected to surgical procedures. On the other hand, active individuals who reveal pronounced weakness in sustaining abduction against resistance or who are unable to sustain abduction after the arm is abducted passively should be offered operative relief.

SURGICAL TREATMENT

1 Incomplete Tears. The sole objective of operative intervention in this type of lesion is to allay abnormal friction between the coraco-acromial arch and the musculotendinous cuff. Whereas the continuity of the cuff is not interrupted it is often difficult to identify and localize lesions in the synovial side. As previously recorded in many instances the bursal floor appears relatively normal yet extensive fraying, tearing and shredding of tendon fibers exist on the joint side of the cuff (Fig. 150). McLaughlin described four simple maneuvers which solve this problem.

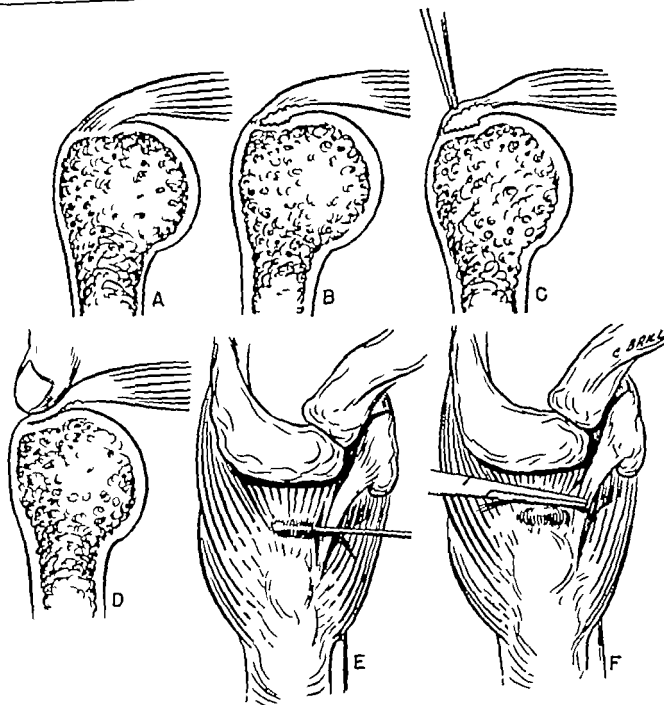


FIG 150 Aids in arriving at a diagnosis of incomplete tears of the cuff (A) Normal insertion of the cuff into the humeral head fibers are firmly attached and cannot be lifted off (B) Rim rent or incomplete tear (C) Outer fibers of cuff in incomplete tears can be lifted from the head of the humerus (D) Finger can palpate sulcus and irregularities on the humeral head through the thinned cuff (E) Longitudinal split in cuff will permit probe to be inserted into the side of the cuff also permitting visualization of the inner surface of the cuff (F) Blister is raised when instrument is passed laterally over the cuff (Redrawn, with modifications from McLaughlin)

1 Normally upon abduction of the arm in various planes of rotation the musculotendinous cuff passes under the coracoacromial arch without a wrinkle on its superficial surface. However, if a rim rent

is present as the arm is abducted a wrinkle or 'blister' forms at the site of the lesion as it approaches the falciform edge of the coracoacromial ligament or edge of the acromion

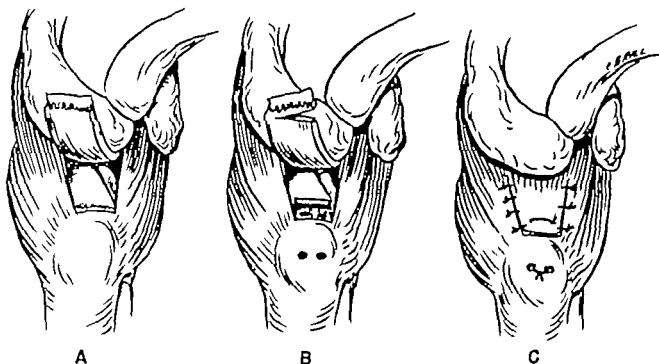


FIG 151 Repair of incomplete ruptures of the supraspinatus region of the cuff (A) A longitudinal incision is made parallel to the tendon fibers between the supraspinatus and the subscapularis tendons next a transverse incision is made extending posteriorly. The length equals the width of the rupture. A second longitudinal incision is made parallel to the first. (B) The affected portion of the cuff is excised. A bony trough is made in the anatomic neck of the humerus. (C) The freshened end of the cuff is reattached by mattress sutures.

2 The blister can be demonstrated by passing a flat instrument laterally toward the tuberosities over the surface of the cuff (Fig 150F)

3 By grasping the affected cuff with a forcep it will be found freely movable on the humerus while the normal cuff on either side fails to show this degree of mobility (Fig 150C)

4 Visualization of the deep surface of the cuff through a small longitudinal incision through its entire thickness is justifiable if the afore-mentioned signs are present (Fig 150E)

A fifth test may be added to the above. Simple palpation of a relatively normal subacromial bursal floor will often disclose thinning of the cuff pronounced irregularity of its deep surface, a sulcus and at times both sulcus and eminence (Fig 150D)

TECHNIC OF REPAIR OF LESIONS ON

THE SYNOVIAL SIDE OF THE CUFF (Mc LAUGHLIN) McLaughlin's transacromial approach provides adequate exposure of the subacromial region. The incomplete tear of the cuff is first visualized by a longitudinal incision through the fibers of the coraco-humeral ligament (between the subscapularis and the supraspinatus muscles). A transverse incision is then made through the whole thickness of the cuff extending posteriorly for a distance equal to the length of the incomplete tear. As a rule the transverse limb of the incision terminates at the juncture of the supraspinatus and the infraspinatus tendons (Fig 151A)

A second longitudinal incision is made beginning at the posterior edge of the transverse incision, paralleling the first (Fig 151B). This mobilizes completely the affected region of the cuff. The degenerated impaired portion of this mobile flap is excised transversely. The remodeled end of

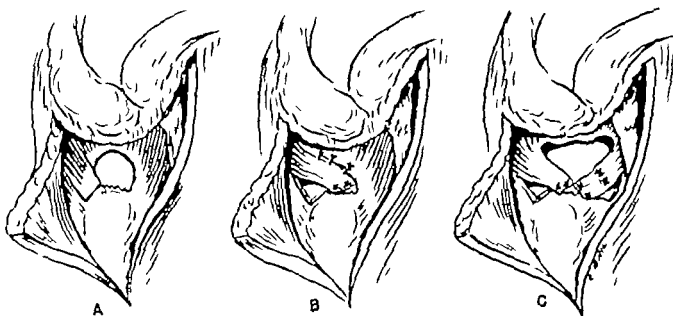


FIG 152 Method of repair of complete ruptures after Lawrence Jones (A) Site of tear (B) Repair effected by restoration of the efficiency of the central link of the cuff by utilizing a single flap cut from the infraspinatus portion of the cuff (C) In more extensive tears 2 flaps are cut, one from the infraspinatus and the other from the subscapularis portion of the cuff. The latter may not be sufficiently long to reach the desired site of reattachment into the greater tuberosity, in which case the tendon is lengthened by a strip of fascia lata.

the cuff should consist of healthy tendon fibers throughout its full thickness. Next, a trough is made in the anatomic neck of the humerus, and the end of the healthy flap is anchored in it by means of mattress sutures passed through holes drilled through the lateral surface of the greater tuberosity. The lateral margins of the flap are attached to the adjacent subscapularis and infraspinatus tendons by interrupted side-to-side sutures (Fig 151C).

Incomplete transverse tears on the bursal side of the cuff are treated similarly to those on the synovial side. However tears within the substance of the cuff or superficial tears running parallel with the cuff fibers can be excised readily by elliptical incisions paralleling the tendon fibers. Closure of the remaining gap is achieved by interrupted side-to-side sutures.

Not infrequently incomplete tears particularly those affecting the bursal side of the cuff, are associated with pronounced secondary inflammatory changes in the subacromial bursa. Such changes, as previously

noted, comprise villous formation on the floor of the bursa, thickening of the synovial walls and adhesions within the bursal sac. In view of these findings recovery will be hastened and become more complete if the bursa is excised. This is done routinely by the author.

Moreover, to preclude impingement of the repaired cuff and greater tuberosity against the acromion and the coraco-acromial ligament, the osteotomized portion of the acromion always is removed, and the coraco-acromial ligament is divided close to its insertion into the medial edge of the acromion.

2. Complete Tears. Numerous methods of repair have been described for complete lesions of the musculotendinous cuff. In general, the rationale of these procedures is based on the concept believed to be responsible for the mechanism of the tear.

Codman advocated end-to-end suture of the torn tendon. If the distal stub of the tendon was absent or frayed so it would not hold a suture the proximal end of the

tendon was sutured into the greater tuberosity. This method was conceived on the premise that all primary tears are transverse tears and that the ultimate triangular or crescentic defect discernible in old cases is produced by retraction of the supraspinatus muscle.

Both Wilson and Bosworth devised methods of repair which were based on Codman's concept. They differ only in the manner of attachment of the torn cuff to the humerus. The method described by McLaughlin is based on an entirely different concept of the mechanism of defect production, while Jones' approach to the problem is based on the functional importance of various components of the musculotendinous cuff.

Jones postulated that shoulder motion is the result of co-ordinated movements of two groups of muscles. One unit, the short rotators, stabilizes the humeral head and the other stabilizes the glenoid. He contended that simultaneous contraction of the capsular muscles resulted in a pull against their bony insertion into the humeral head and also a pull against one another through the medium of the fibrotendinous cuff in which the tendon fibers of all muscles are welded into one continuous sheath. The central link of the various components of the cuff is the supraspinatus muscle, whose weight is approximately one-seventh of the total weight of the four short rotator muscles. In view of the relative weakness of this muscle, Jones believed that its interruption in complete cuff lesions was not responsible for the loss of abduction. He was also of the opinion that interruption of the central link of the conjoint cuff reduced the mechanical efficiency of the subscapularis muscle anteriorly and of the infraspinatus and the teres minor muscles posteriorly. Jones' procedure therefore tends to restore the functional continuity of the cuff but disregards the supraspinatus muscle (Fig. 152).

McLaughlin contends that the defect noted in complete tears of the cuff is a combination of a transverse tear and a longitudinal split through the cuff usually start-

ing at the anterior edge of the transverse tear. Divergent forces of the subscapularis muscle, pulling anteriorly (and of the remaining rotators, pulling posteriorly), are responsible for the triangular or crescentic defect in the cuff (Fig. 137). His method of repair attempts to restore the anatomic continuity and the mechanical efficiency of the cuff. The high percentage of excellent results obtained with this procedure substantiates McLaughlin's concept and justifies his method.

TECHNIC (McLAUGHLIN) The essential features are excision of the peripheral margins of the defect to healthy vascular tissue and reduction of the defect by side-to-side repair up to but not beyond the point of tension and reinsertion of the retracted portion of the cuff into the humeral head, at whatever point it will reach without tension when the arm is at the side. Although the technic varies in each case, the aforementioned principles are applicable to all degrees of tears with retraction ranging from tears with only slight retraction to massive avulsion of the cuff with marked retraction as far as the level of the glenoid.

The subacromial region is exposed through the transacromial incision. An oblique osteotomy is done through the acromion beginning anteriorly at a point midway between the acromioclavicular joint and the lateral margin of the acromion and terminating at the lateral tip of the acromion. Having defined the limits and the character of the tear, its peripheral degenerated fibrotic margins are excised back to healthy vascular tendon tissue. Generally, the tear is converted into a V-shaped defect whose apex points medially (Fig. 153A).

A shoelace type of continuous suture (No. 5 Deknatel) is employed, beginning at the apex of the opening. Traction on the ends of the suture approximate the adjacent margins of the defect. Suturing is continued in this fashion up to the point of tension—never beyond it—and so only a small V-shaped hiatus remains. Next the articular cartilage immediately below the remaining

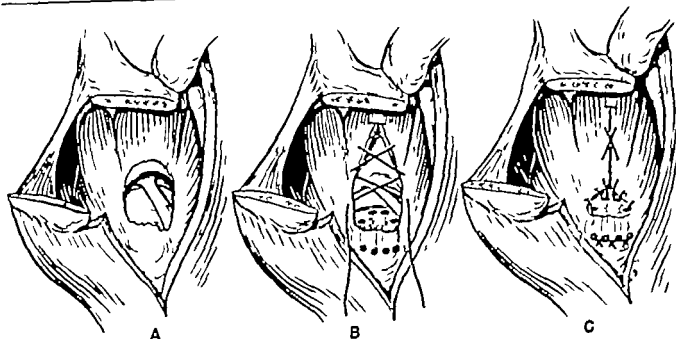


FIG 153 Method of repair of complete ruptures (after McLaughlin) (A) Rupture in cuff exposed through a transacromial incision the degenerated margins of the defect are excised back into healthy tissue. (B) Shoelace type of continuous suture beginning at the apex of the defect is used to approximate the freshened edges of the cuff (C) The edges are approximated up to the point of tension with the arm at the side a triangular hiatus usually remains in the cuff The articular cartilage of the head of the humerus just below the defect is removed, exposing raw bone. The cuff is reattached to the humerus by passing the ends of the continuous suture through drill holes, tying them on the outer surface of the greater tuberosity If necessary, additional mattress sutures may be added to attain better approximation of the edges of the cuff to raw bone

defect is removed exposing the raw subchondral bone. Enough cartilage is removed to place the edges of the defect in contact with raw bone. The two continuous sutures are then passed through drill holes and tied onto the outer surface of the greater tuberosity. Occasionally additional mattress sutures may be necessary to secure the edges of the defect firmly against the new bony insertion (Fig 153B and C).

It is advantageous to discard the osteotomized portion of the acromion to divide the coraco-acromial ligament at its insertion into the medial edge of the acromion and, finally to excise as much bursal tissue as possible. This favors the passing of the tuberosity and the musculotendinous cuff under the coraco-acromial arch without impingement and undue friction. The deltoid muscle is sutured to the fascia and the periosteum over the remaining portion of the acromion. No appreciable loss of deltoid

efficiency has been demonstrable in cases so treated.

POSTOPERATIVE MANAGEMENT This phase of the treatment is guided by the severity of the lesion and the willingness and the co-operation demonstrated by the patient. Postoperative management must be supervised *at all times* by the surgeon in harmony with his knowledge of the lesion prior to repair and the type of repair which provide him with valuable guides to dictate how intense and rapid a restoration program may be instituted.

Immediately following operation the arm is placed in balanced suspension until wound healing is completed (Fig 261). Gentle motion is allowed during this period. After the wound is healed the patient is allowed out of bed and is started on a well regulated supervised program of gravity free pendulum exercises to be performed within the tolerance of pain—never beyond

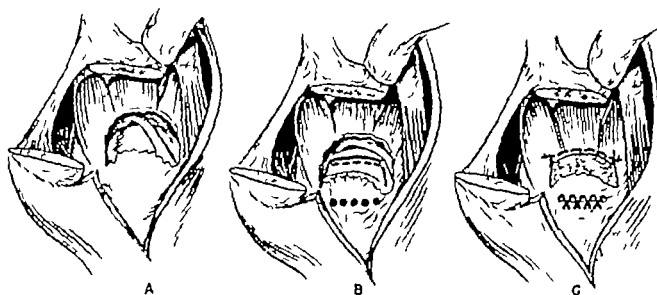


FIG. 154 Repair of massive avulsion of the cuff with advanced retraction (after McLaughlin) Approximation of the lateral edges of the defect may not be possible, freshened edge of the cuff is then attached to the humeral head at whatever point it reaches without tension (A) The transacromial incision adequately exposes the defect in the cuff (B) The edges of the cuff are freshened back to healthy tissue and the articular cartilage is removed from the head of the humerus to provide a raw bony bed. (C) The cuff is reattached to the raw bony surface by mattress sutures passed through drill holes and tied in the outer surface of the greater tuberosity

the point of fatigue Exercises should be performed for short periods (5 minutes) at frequent intervals (every hour on the hour) As pain subsides and strength returns active exercises are increased and wall-crawling exercises are added Active elevation of the arm is never permitted before three weeks. Usually it is safe to allow active elevation between the third and the seventh weeks depending upon the severity of the lesion and the security of the repair Resumption of light normal use is encouraged at once but its rate of increase always should be guided by the above factors

3 Massive Avulsion of the Cuff These lesions are treated by the technic described previously for complete tears with retractions The cuff in most instances can be secured at some point to the humeral head thereby providing a suspensory support for the head and also improving the efficiency of the rotator muscles Generally restoration of function is sufficient to justify the procedure. Rarely is it necessary to resort to arthrodesis of the glenohumeral joint In

only one instance in which there was complete avulsion of the cuff which had retracted beyond the glenoid cavity was the author forced to do an arthrodesis In this case it was impossible to mobilize any part sufficiently to reinsert it into the humeral head Usually however while the cuff in massive avulsions lies between the glenoid and the humeral head it can be mobilized sufficiently to allow reinsertion into the head (Fig 154)

Theoretically a Nicola procedure in addition to the cuff repair should add further stability of the humeral head in cases of massive avulsions of the cuff However the author never has felt the need of this additional procedure None of the thirteen cases of massive avulsion of the cuff treated by McLaughlin's method showed any tendency toward subluxation of the humeral head.

4 Concomitant Lesion with Incomplete and Complete Tears of the Cuff

BICIPITAL TENOSYNOVITIS. The incidence of bicipital tenosynovitis accompanying tears of the musculotendinous cuff is greater



FIG 155 Multiple areas of ossification in the musculotendinous cuff of a male 26 years of age. The individual dislocated the shoulder 3 years prior to the taking of this roentgenogram. At operation more heterotopic bone was demonstrable than the roentgenogram depicts. Note the scalloped defect in the superolateral aspect of the head of the humerus. It was impossible to excise the bone without removal of the greater portion of the cuff thereby seriously impairing the rotator and stabilizing mechanism of the glenohumeral joint.

than is generally realized. It may be caused by the initial trauma or by secondary inflammatory changes in the bicipital sulcus and the impaired portion of the musculotendinous cuff bridging the groove. Pain and stiffness persist in many instances of cuff injuries, after the acute phase has subsided. It has been the author's experience that in the majority of cases, the disability is not caused by an incomplete or mild complete tear of the cuff but by a bicipital tenosynovitis. The nature of the syndrome varies with the severity of the lesion. This observation has been confirmed at operation many times and is discussed fully in the chapter dealing with bicipital syndromes.

TENDINITIS OF ROTATOR TENDONS. Existing degenerative alterations in the cuff past middle age along with the inflammatory processes that are discernible after incomplete or complete tears of the cuff force one to concede that tendinitis does exist and that it may give rise to symptoms. However, it has been proved conclusively that lesions of the cuff which do not materially impair the normal mechanics of the shoulder joint do not necessarily produce symptoms. Symptoms are manifest only when there is an actual impediment to

smooth joint motion, such as impingement of the torn cuff and the greater tuberosity against the acromion or the coraco-acromial ligament during abduction or elevation of the arm. It is obvious that tendinitis, per se is not the responsible lesion for the syndrome. Mechanical obstructions to normal motion are the true etiologic factors. Restriction of the normal arcs of motion, a characteristic feature of these lesions is responsible for vascular and lymphatic stasis, which, in turn are followed by fibrosis, contracture and shortening of the capsular elements, shortening of the rotator muscles and shortening adductor muscles of the shoulder. The biceps tendon and sheath are also involved in this process and further restrict the joint motion. The clinical picture is now one of frozen shoulder. It is interesting to note that a frozen shoulder never has been observed in association with a complete cuff tear. Tendinitis is considered in more detail in the chapter on frozen shoulder and bicipital syndromes.

Calcareous deposits are occasionally demonstrable in torn degenerated cuff fibers and osseous tissue may be discernible in the cuff in rare instances. These observations were also noted in several cadaver and post



FIG 156 Heterotopic bone formation in the cuff. An acromioplasty was done and the extremity was treated with a sling followed by early mobilization.



FIG 157 Hypertrophied subacromial bursa removed from the shoulder of an individual exhibiting a typical painful arc syndrome. Exploration of the inner aspect of the cuff disclosed also an incomplete tear in the supraspinatus region which was repaired. The patient made an excellent recovery.

mortem specimens. Such alterations are indicative of pronounced degenerative changes in the tendon fibers (Fig 155 and 156).

BURSITIS. Subacromial bursitis does not exist as a primary entity. It is always secondary to some other pathologic change in the adjacent tissues. The close proximity of the floor of the bursa to the superficial surface of the musculotendinous cuff renders it vulnerable to lesions involving this region. In all cases of complete tears of the cuff in the supraspinatus region and incomplete tears of the superficial fibers of this portion of the cuff the floor of the bursa reveals some degree of involvement. Severity of the alterations in the bursa is as a rule in direct ratio to the extent of the cuff impairment. Once established bursal changes which comprise villi adhesions and thickening of the bursal walls will add further hindrance to joint movements. The bursal tissues are richly supplied with sensory nerve fibers. Impingement of these tissues against the coraco-acromial arch elicits pain, thereby restricting motion. On

the basis of these observations it is justifiable to excise as much of the bursa as possible in surgical procedures in this region (Fig 157).

EXOSTOSES. Exostoses may form on the tip of the greater tuberosity at the insertion of the supraspinatus tendon. They may be concomitant lesions with a cuff tear or may occur following fracture of the tuberosity without tearing of the cuff (Fig 141). Bosworth reported an instance following sudden hyperextension of the arm. Impingement of the bony mass against the acromion or the coracohumeral ligament elicits pain and a jog. Radiographic examination in different positions will disclose the lesion. Excision of the exostoses is essential in order to relieve distressing symptoms.

TEARS CONCOMITANT WITH DISLOCATIONS, FRACTURES AND FRACTURE-DISLOCATIONS OF THE HUMERAL HEAD. Rupture of the cuff is not an infrequent lesion accompanying dislocations, fractures and fracture-dislocations of the humeral head. They are discussed fully in the chapter dealing with these traumatic lesions.

5. ACROMIOPLASTY. There is now general agreement that removal of the lateral por-

tion of the acromion has no harmful effects. It should be done routinely in all repairs for both complete and incomplete cuff tears. Removal of portions of the acromion eliminates all abnormal impingement of the impinged cuff against the acromion and minimizes normal friction. Therefore, the procedure favors early restoration of joint function. McLaughlin describes two types of osteotomies through the acromion: (1) an oblique osteotomy which starts anteriorly, midway between the acromioclavicular joint and the lateral margin of the acromion and extends laterally to emerge at the tip of the acromion and (2) an anteroposterior osteotomy which starts at the

same point anteriorly as the oblique osteotomy and extends directly posteriorly (Fig. 338). The latter is utilized to obtain maximum exposure of the cuff, whereas the former gives slightly less exposure but a better cosmetic result.

The deltoid muscle which is stripped from the lateral margin of the acromion is reattached to the fascia and the periosteum over the remaining medial portion of the acromion. Deltoid power is not impaired by this procedure.

If deemed necessary the entire acromion may be removed without ill effect, provided that the coracoclavicular ligaments are intact.

BIBLIOGRAPHY

- Bosworth, D. M. An analysis of twenty-eight consecutive cases of incapacitating shoulder lesions radically explored and repaired, *J. Bone & Joint Surg.* 22: 369-392, 1940.
- Codman, E. A. *The Shoulder*. Boston: Thomas Todd Co., 1934.
- Duchenne, C. B. *Physiologie des mouvements*. Paris, Bailière, 1867, pp. 7-17.
- Inman, V. T., Saunders, J. B., deC. M., and Abbott, L. C. The function of the shoulder joint, *J. Bone & Joint Surg.* 26: 1-30, 1944.
- Jones, L. The shoulder joint: observation on the anatomy and physiology with an analysis of a reconstructive operation following extensive injury. *Surg. Gynec. & Obst.* 75: 433, 1942.
- Lippman, R. K. Frozen shoulder periarthritis bicipital tenosynovitis. *Arch. Surg.* 47: 283, 1943.
- Lippman, R. K. Bicipital tenosynovitis, *New York State J. Med.* 44: 2235-2240, 1944.
- McLaughlin, H. L. Lesions of the musculotendinous cuff of the shoulder. *J. Bone & Joint Surg.* 26: 31-51, 1944.
- Meyer, A. W. Unrecognized occupational destruction of the tendon of the long head of the biceps brachii. *Arch. Surg.* 2: 130-144, 1921.
- Meyer, A. W. Spontaneous dislocation of the long head of biceps brachii. *Arch. Surg.* 13: 109-119, 1926.
- Meyer, A. W. Spontaneous dislocation and destruction of the tendon of the long head of biceps brachii. *Arch. Surg.* 17: 493-506, 1928.
- Meyer, A. W. Chronic functional lesions of the shoulder. *Arch. Surg.* 35: 646-674, 1937.
- Mosely, H. F. *Shoulder Lesions*. Springfield, Ill., Thomas, 1945.
- Outland, T. A. M., and Shephard, W. F. Tears of the supraspinatus tendon, résumé of twelve operated cases, *Ann. Surg.* 107: 116-121, 1938.
- Stevens, J. H. The action of the short rotators on the normal abduction of the arm with a consideration of their action in some cases of subacromial bursitis and allied conditions. *Am. J. M. Sc.* 138: 870, 1909.
- Wilson, P. D. Complete rupture of the supraspinatus tendon, *J.A.M.A.* 96: 433-439, 1931.

Frozen Shoulder and Bicipital Syndromes

FROZEN SHOULDER

BICIPITAL TENOSYNOVITIS

BICIPITAL TENOSYNOVITIS

ASSOCIATED WITH TRAUMA

BICIPITAL TENOSYNOVITIS WITHOUT TRAUMA

CORACO-ACROMIAL LIGAMENT

CORACOHUMERAL LIGAMENT

RUPTURE OF THE BICEPS TENDON

PROXIMAL RUPTURES

TRAUMATIC DISLOCATION

HISTORICAL SURVEY

The enigma of the "painful stiff shoulder" has been the source of much speculation by workers interested in this problem. Many and varied pathologic lesions have been recorded in the literature as probable etiologic factors. Nevertheless there is a general feeling that the true causative agents of this syndrome and the developmental steps culminating in partial or complete loss of scapulohumeral motion as yet have not been uncovered.

Duplay (1896) was the first investigator to focus our attention on the extra-articular tissues of the scapulohumeral joint as a possible source of pain and limitation of motion. He was of the opinion that inflammation of the subacromial bursa was the responsible pathologic lesion for the above clinical features and coined the term "scapulohumeral periarthritus." The medical profession quickly adopted this term which soon came to embrace a large and heterogeneous group of cases whose only common denominators were pain and stiffness in the region of the shoulder joint. It is astonishing to note with what reluctance the term "periarthritus" is being discarded for it provides a designation for a host of unexplainable shoulder lesions.

Calcareous deposits in the rotator cuff

chiefly in the supraspinatus tendon were finally recognized as capable of producing this syndrome. As the result of the meticulous and comprehensive studies of Painter (1907), Baer (1907), King and Holmes (1927), Mumford and Martin (1931) and Dickson and Crosby (1932) this group of cases was taken out of the heterogeneous classification of "periarthritus" and became recognized as an unequivocal clinical entity. However credit must be given to Codman (1934) for the clarification of the pathogenesis of this entity. He also set forth the principles of therapy which we employ at the present time. Codman demonstrated that the lesion developed primarily in the degenerated fibers of the fibrotendinous cuff and that symptoms arose only when the synovial lining of the subacromial bursa became involved. These observations have been verified many times and now there is general agreement on this point.

Calcareous tendinitis is responsible for pain and stiffness in the scapulohumeral joint in approximately 50 per cent of the individuals with painful and stiff shoulders. (McLaughlin 1944). It becomes apparent therefore that a host of individuals remained without adequate explanation of their malady. Again credit goes to Codman who extracted from this residue another group of cases. His exhaustive studies on the

rotator cuff of the shoulder joint gave us another undisputed, crystallized clinical entity—rupture of the supraspinatus tendon—which is now recognized as a common causative agent of this symptom complex.

In spite of these contributions, there remained still a large number of cases which had neither calcareous deposits in the fibro-tendinous cuffs nor rupture of the supraspinatus tendons. Meyer (1921, 1926, 1928 and 1930) was the first observer to point to the long head of the biceps brachii muscle as a possible source of disorders about the shoulder joint. These studies were made on shoulder joints of cadavers. He described degenerative and attritional lesions comprising fraying, shredding, fasciculations and tearing of the fibers of the biceps tendon. Some specimens exhibited partial or complete dislocation of the tendon out of the bicipital groove. In several shoulder joints the intracapsular portion of the tendon was absent while the proximal end of the extracapsular portion had attained a bony insertion in the region of the lesser tuberosity. He attributed these lesions to the following causes: (1) contact of the tendon with a supra-tubercular ridge when present, (2) roughening and irregularities of the floor of the sulcus and (3) contact of the tendon with cartilaginous margins of the humeral head or with the lesser tuberosity in those instances of partial or complete dislocation. He was of the opinion, furthermore, that these alterations were the result of using the upper extremity in a position of abduction and external rotation.

Gilcreest (1926) provided clinical data in support of rupture of the long head of the biceps tendon. In a comprehensive investigation of 100 cases he recognized an "acute" and a "latent" rupture of this structure and recorded that the "latent" group was often unrecognized and erroneously diagnosed as bursitis, arthritis, sprain, etc. This work was followed by another study published in 1936 on Spontaneous Dislocation of the Tendon of the Long Head of the Biceps Brachii.

Although vague references had been made to tenosynovitis of the long head of the biceps brachii muscle, both in this country and abroad, it was not until 1932 that Pasteur (1933) correlated the observations of Meyer and the clinical picture characterized by pain and stiffness in the shoulder joint. He described the syndrome, teno-bursitis, as a new clinical entity and believed it to be the responsible pathologic disorder underlying periarthralgias and ankylosis of the shoulder joint. Pasteur was the first observer to associate tenosynovitis of the long head of the biceps brachii muscle with the 'frozen shoulder'. Schragar confirmed Pasteur's observations and described, for the first time in American literature, tenosynovitis as a definite clinical entity. These contributions were followed by many comprehensive articles in American literature, all of which emphasized the close relationship between lesions of the biceps tendon and the syndrome characterized by pain and stiffness in the shoulder joint (Abbott, L. C., and Saunders, J. B.; Tarsy, J. M.; Mosely, H. F.; Hitchcock, H. H., and Bechtol, C. O.).

Lippmann (1944) described adhesive tenosynovitis as the cause of 'frozen shoulder'. He reported twelve cases which, on exploration, exhibited an inflammatory process of the long head of the biceps and its sheath with no involvement of any of the periarthral structures. Although he did not demonstrate the lesion, he was of the opinion that the limitation of motion was due to extension of the inflammatory process, involving the biceps tendon into the joint, producing adhesions which in turn fixed the intracapsular portion of the tendon to the capsule and the articular surface of the humerus. He recorded that upon releasing the peritendinous adhesions, full motion was restored readily without forceful manipulation.

Codman was of the opinion that the biceps tendon and its sheath played only a minor role if any in the production of shoulder symptoms. He wrote, "Personally, I believe that the sheath of the biceps tendon is less

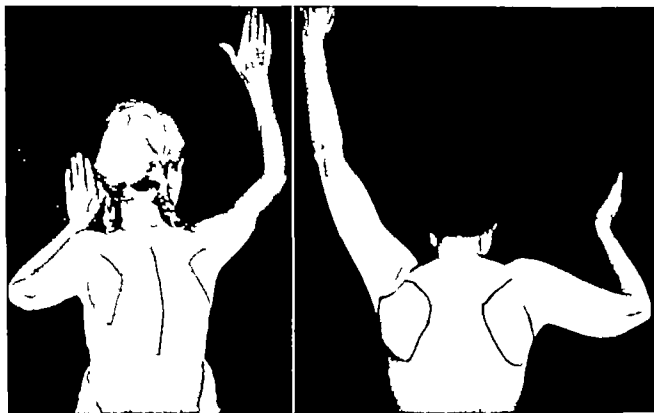


FIG 158 (*Left*) Characteristic features of a patient with a left frozen shoulder. The patient's general health is below par—she has been a victim of bronchiectasis for many years. Note the relation of the axis of the shaft of the humerus to that of the spine of the scapula forming approximately a right angle. This relationship is maintained during all movements of the arm and is more clearly discernible in roentgenograms taken during different stages of elevation of the arm.

FIG 159 (*Right*) Same characteristic features as those pointed out in Figure 158 except that the patient's general health is better. Roentgenograms of both extremities were made during different stages of elevation (Figs. 160-161).

apt to be involved than are the other structures. I have never proved its involvement in a single case. I think that the substance of the tendon of the supraspinatus is the most often involved. He believed that the pathology of frozen shoulder was essentially tendinitis of the rotator tendons.

From the above survey it is obvious that much work remains to be done before clarification of this symptom complex is attained.

FROZEN SHOULDER

INTRODUCTION

The observations recorded in this chapter referable to frozen shoulder and bicipital tenosynovitis were made on a clinical investigation of 83 cases of which 56 were

investigated surgically. Correlation of these findings with those noted in the study on degenerative lesions of the shoulder joint permitted one to make some logical deductions referable to the causes and the pathogenesis of frozen shoulders.

CLINICAL FEATURES

Frozen shoulder is a definite clinical entity. There is a characteristic cycle of events. It usually occurs in individuals after 40 years of age (only 1 case in this series was under 40); the greatest number are found between 50 and 60 years; more women (67 per cent) than men are affected and it is a relatively common disorder in patients afflicted with cardiovascular, pulmonary or metabolic diseases (Fig 158).



FIG 160 Roentgenographic studies of a frozen shoulder, showing the relation of the axis of the shaft of the humerus to the spine of the scapula in different stages of elevation of arm. Note (top right and bottom) some elevation of the humerus in relation to the trunk has been achieved over the position of the arm (top left) how ever little or no change has occurred in the relation of the axis of the humerus to that of the spine of the scapula, indicating that the movement has occurred in the thoracoscapular joint and not in the glenohumeral articulation. Compare with Figure 161

The syndrome may develop after some form of injury to the shoulder on the other hand the onset may be insidious without injury. In the early phase of the disease there are varying degrees of pain and stiff ness in the shoulder. Increased activity accentuates the pain which is usually localized over the anterolateral aspect of the shoulder. Frequently, the pain radiates to the anterior aspect of the middle of the upper arm and occasionally to the flexor surface of the forearm. A common complaint is pain during the night which frequently interferes with sleep. Tenderness always can be elicited by making pressure over the bicipital groove or rolling the biceps tendon under the exam-

iner's thumb. There is a steady increase in the severity of the symptoms while the arcs of painless motion in the glenohumeral joint become progressively smaller until there is little or no motion in the joint. Both the deltoid and the spinatii muscles exhibit varying degrees of spasm and atrophy. The extremity is held at the side in a position of internal rotation. Generally a few degrees of free painless motion are demonstrable in the glenohumeral joint in the anteroposterior plane. Occasionally, compression of the belly of the biceps brachii muscle evokes pain. Not infrequently all the cardinal signs and symptoms of a scale nus anticus syndrome are present and may

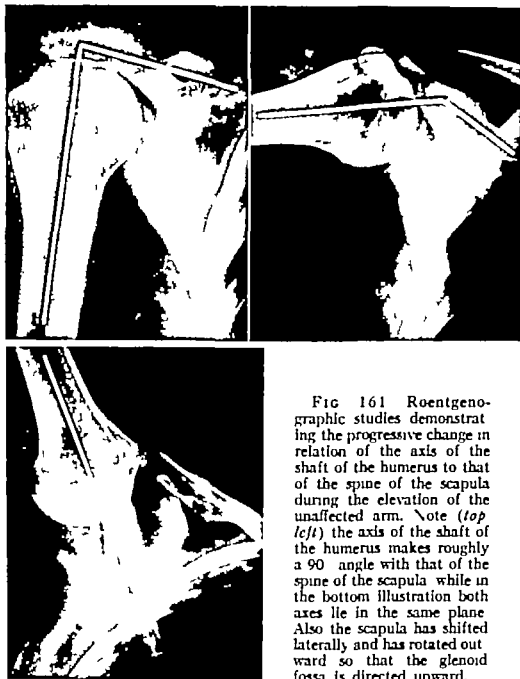


FIG 161 Roentgenographic studies demonstrating the progressive change in relation of the axis of the shaft of the humerus to that of the spine of the scapula during the elevation of the unaffected arm. Note (*top left*) the axis of the shaft of the humerus makes roughly a 90° angle with that of the spine of the scapula while in the bottom illustration both axes lie in the same plane. Also the scapula has shifted laterally and has rotated outward so that the glenoid fossa is directed upward.

confuse the clinical picture. Clinically there now exists a fibrous ankylosis of the scapulohumeral joint. It is interesting to note that in some instances when all motion is lost pain may become bearable and in fact may disappear in other instances there is intractable pain (Figs 160 and 161).

At this point it must be pointed out that pain is the most significant feature of the syndrome. Although it is true that there exists pronounced restriction of movements in the scapulohumeral joint most patients

ascribe their disability to the pain rather than to loss of movements.

The clinical course which frozen shoulders pursue is variable and unpredictable because it may terminate at any stage of its development or remain static for months and even years before regression of the syndrome begins. In many instances after a variable period of time pain subsides and slow restoration of function begins. A normal shoulder joint is not regained in all instances; many individuals exhibit per-

manent weakness and some residual loss of movements in all directions in the scapulohumeral joint. As stated in many articles on the subject, it is erroneous to believe that complete and spontaneous recovery within 6 months to 3 years always occurs. Three patients in this series exhibited painful stiff shoulders for 5, 6 and 8 years, respectively, with no indication of regression of the malady.

ETIOLOGY AND PATHOGENESIS

- ① Muscular inactivity is the causative agent responsible for frozen shoulder. Any cause which restricts scapulohumeral movement produces muscular inactivity. Bicipital tenosynovitis, as will be shown subsequently, is the most common etiologic factor, some other causes are contusions sprains dislocations, fracture of the shoulder, and injuries to the musculotendinous cuff. Restriction of scapulohumeral motion coincident to treatment of injuries of the upper extremity at a distance from the region of the shoulder as fractures of the forearm and wrist, also produces inactivity of the shoulder musculature which may terminate in a frozen shoulder.

As a result of functional inactivity, regardless of the cause muscle tone is diminished, and muscle atrophy supervenes. The normal delicate balance of the metabolic processes of the tissues is disturbed and there follows a slowing down of the circulation and venous and lymphatic stasis. All soft tissues adjacent to and comprising the musculotendinous cuff become saturated with serofibrinous exudates which provide the fibrin responsible for formation of capsular synovial, fascial intermuscular and intramuscular adhesions. A low grade chronic inflammatory process ensues which involves all the tissues affected. The process begins in all tissues simultaneously, and not in one specific area. Findings consistent with a chronic inflammatory process are readily discernible in microscopic sections made from the different areas of the musculotendinous cuff the biceps tendon and

the subacromial bursa (Fig 162). On the other hand, functional activity reverses the above process and restores circulation and joint function.

The shoulder joint is unique in that its anatomy renders it susceptible to changes which produce stiffness. Its articular capsule is loose and redundant, when the arm is in the sling position numerous nictitating folds are formed in the inferior and anterior aspects of the glenohumeral joint (Fig 15). Moreover, as the short rotator muscles cross the capsule to their respective points of insertion in the head of the humerus, they provide large surface areas on which serofibrinous fluid may accumulate. This is also true of the large surface areas between the outer and the inner muscular sleeves (the deltoid forms the outer sleeve, the spinatus and the subscapularis muscles the inner sleeve). Finally, the subscapularis recesses and subacromial bursa are suitable sites for accumulation of serous exudates.

Frozen shoulders develop only when the afore-mentioned phenomenon occurs in shoulder joints exhibiting profound degenerative alterations in the synovials, the musculotendinous cuff and the biceps tendon. In the presence of these changes the process progresses until there is firm fixation of the scapulohumeral joint by extra-articular and intracapsular adhesions.¹ All structures lose their elasticity and those on the anterior aspect of the glenohumeral joint become shortened. It was interesting to observe the effect of these changes on the coracohumeral ligament. It is converted into a tough, inelastic contracted cord of fibrous tissue extending from the coracoid process to both tuberosities of the head of the humerus (Fig 18). This structure, together with the shortened subscapularis muscle, functions as strong checkreins to

¹ The term "intracapsular adhesions" is used to designate adhesions between the nictitating folds of the synovials, particularly in the anterior and inferior aspects of the joint and in those binding the synovial side of the capsule to the inferior surface of the head of the humerus.



FIG 162 Degenerative and inflammatory changes in all the soft tissue components of the scapulohumeral articulation of an individual with frozen shoulder. Microscopic study reveals edema of the tissues, degeneration of the collagenous fibers, pronounced round-cell infiltration of the tissues, increased vascularity, marked thickening of the synovial membrane and evidence of increased fibrosis. These findings are consistent with a low grade chronic inflammatory process superimposed on degenerative alterations in the soft tissue elements affected. (Top left) A microscopic section through the walls of the subacromial bursa ($\times 245$). (Top right) Microscopic section through the coracohumeral ligament ($\times 245$). (Bottom left) Microscopic section through the supraspinatus region of the musculotendinous cuff ($\times 245$). (Bottom right) Microscopic section through the subscapularis tendon ($\times 245$).



FIG 162 (Continued) Microscopic section through the biceps tendon. Note the marked thickening of the synovial membrane and increased cellular elements in this tissue ($\times 250$)

external rotation, while the infraspinatus and the teres minor muscles restrict internal rotation. All cases explored demonstrated varying degrees of involvement of the tendon tendon-sheath gliding mechanism of the biceps tendon. Bicipital tenosynovitis is a concomitant lesion of all frozen shoulders, its implication may be primary or secondary.

It has been demonstrated amply that gross degenerative lesions occur in all individuals after the fourth decade that microscopic lesions occur after the third decade that these alterations increase in intensity with each subsequent decade and finally that in the later decades they may assume profound proportions (Fig 66). This explains the occurrence of frozen shoulders in individuals past 40 years of age and its rarity before this age period. Moreover constitutional states which tend to increase the severity of degenerative alterations in tissues render patients afflicted with such

disorders still more vulnerable. It is common knowledge that frozen shoulders are encountered frequently in debilitating disorders as cardiac, pulmonary and metabolic diseases.

Bicipital tenosynovitis plays a major role in the symptomatology and the course pursued by frozen shoulders. Involvement of the tendon tendon sheath gliding mechanism may be primary or secondary nevertheless, regardless of its origin, once it is established it gives rise to pain in the shoulder, especially when the extremity is put through certain arcs of motion. Because of the resulting pain, further functional activity results, a situation which favors circulatory impairment in the soft tissues and formation of adhesions. The location of the pain over the bicipital groove and tenderness elicited on pressure over this area and over the biceps tendon forces one to conclude that bicipital tenosynovitis is the prime causative factor responsible for pain both in the early and in the late phases of the disease. This has been proved many times in cases in this series, in which, after excision of the intracapsular portion of the tendon and transplantation of the proximal end of the extracapsular portion to the coracoid process, there was dramatic and instantaneous relief of pain.

Clinical assessment reveals that in many cases of frozen shoulders after a period of pain and disability, pain disappears, and shoulder function is slowly regained. It is reasonable to assume that in these cases the inflammatory process subsides, muscle spasm is relieved, adhesions undergo resolution, and muscular activity is restored, which in turn tends to restore normal circulation to the affected tissues. In other instances pain and stiffness persist. Pain is the outstanding feature and results from involvement of the biceps tendon and the tendon sheath. A cure can be effected in these cases only by obliteration of the tendon tendon sheath mechanism; nature achieves this in some instances by anchoring the tendon to the shaft of the humerus be-

low the lesser tuberosity. By so doing the pain factor is eliminated, and restoration of joint function can begin. Such a clinical course is illustrated by Case J F (p. 109). In still another and smaller group of cases nature fails to eliminate the biceps tendon gliding mechanism, therefore, spontaneous recovery does not occur, and pain and stiffness persist.

It appears that the ultimate result depends directly on the degree of degenerative alterations existing in the structure comprising the scapulohumeral joint. This is borne out by the clinical observation that in individuals under 40 years of age in whom only minimal changes are observed and in whom the circulation of the tissue is excellent, frozen shoulders rarely occur following muscular inactivity in individuals of later decades who are in good general health. Frozen shoulders develop, but spontaneous recovery occurs in most of them (Case M W). However in individuals past middle life afflicted by some debilitating disease which tends to accentuate physiologic degenerative lesions, protracted pain and shoulder dysfunction are the rule (Case T R.).

TREATMENT

Successful management is governed by careful selection of cases and by instituting the correct therapy for each case. Whenever possible the causative or initiating factor responsible for the frozen shoulder should be determined. In many instances elimination of this cause will promote relief of muscle spasm, rapid return of normal muscular activity and restoration of joint function. For example, calcareous tendinitis may be the underlying cause; in such instances simple excision of the pathologic irritative material will start the sequence of events which will lead to recovery. Also bicipital tenosynovitis may be the cause (Case W S). Treatment directed toward effecting a cure of this lesion in turn will alleviate pain and initiate restoration of scapulohumeral motion.

EARLY STAGES OF THE DISEASE

Pain and pronounced muscle spasm are the principal features of this stage.¹ If possible these individuals are best treated by complete bed rest, sedatives to relieve pain, continuous hot fomentations to the shoulder and active exercises in the supine position within the painless arcs of motion. Blocking of the cervical sympathetic ganglia with 10 or 15 cc. of 1 per cent procaine provides considerable comfort and relieves muscle spasm. After from 7 to 10 days the arm is rested in a sling, but pendulum exercises are encouraged on a prescribed time schedule. For example, the arm is exercised every hour, each exercise being done 10 or 12 times always within the painless arcs of motion (Fig. 238). Later, the sling is discarded and crawling up-the-wall and pulley exercises are added. Such a regimen will produce surprisingly excellent results in a goodly number of patients within 8 to 10 weeks. In patients who refuse a period of bed rest, repeated blocking of the cervical sympathetic ganglia (the procedure is done 3 or 4 times every 4 or 5 days) is a valuable adjunct to the therapy described above. Even in the face of a well planned and well executed regimen many cases progress to the later stage.

LATER STAGE OF THE DISEASE

In the later stage pain is usually less and loss of scapulohumeral motion is caused by intracapsular and extracapsular adhesions, shortening of the rotator and the adductor muscles and muscle spasm. Pain and adhesions limit the range of motion to a few degrees (10° to 20°) in the anteroposterior plane. Beyond this arc pain is accentuated. All cases explored in this study disclosed varying degrees of involvement of the biceps tendon gliding mechanism. In some

¹ With complete muscular relaxation under anesthesia these shoulders disclose considerable freedom of motion in the scapulohumeral joint, indicating that muscle spasm (and not adhesions and soft tissue contractions) is responsible for the restricted range of movements.

the tendon was bound down by firm dense adhesions, in others it was enmeshed in a network of filmy adhesions. These findings conform to those of Hitchcock and Bechtol. The author is of the opinion that in these cases recovery is retarded because of (1) the pain factor which precludes motion of the glenohumeral joint beyond a certain restricted arc and (2) the mechanical obstruction to movements provided by adhesions implicating the biceps tendon. Recovery can be hastened and the patient rehabilitated by eliminating the above two factors. This is done simply by severing the biceps tendon at the supraglenoid tuberosity and transplanting it to the coracoid process. Hitchcock and Bechtol first advocated removing the intra articular portion of the tendon. The procedure herein described differs from the one described by these workers only in that the tendon is anchored to the coracoid process instead of to the shaft of the humerus.

OPERATIVE TECHNIC

(See Treatment of Bicipital Tenosynovitis)

MANIPULATION FOR FROZEN SHOULDERS

Manipulation is mentioned only in order to condemn it. In spite of the fact that many investigators have pointed out repeatedly the futility and the dangers of this procedure many victims are still being subjected to the maneuver. As the result of some striking observations noted under direct vision while manipulation procedures were carried out one is forced to conclude that gentle manipulation is impossible for the end results are invariably the same.

In 8 patients, the affected shoulder joint was exposed through a splitting incision of the anterior deltoid. In all instances upon external rotation of the extremity tears of varying degrees were noted in the substance of the subscapularis tendon and its lower muscle fibers inserting into the humeral shaft. In 2 individuals the subscapularis tendon was completely avulsed from the lesser tuberosity and the tear extended

proximally into the fibrotendinous cuff for 1 and 1½ inches, respectively. Upon abduction of the arm, similar transverse tears were observed in the fibrous capsule along the inferior aspect of the humeral neck. In 3 instances, the inferior articular surface of the humeral head came into view through the tears in the capsule. One individual, in whom a manipulation had been done 3 months prior to the procedure done under direct vision, sustained a fracture through the surgical neck of the humerus. Such observations serve to emphasize the hopelessness of such a procedure. In this series 7 patients had had manipulative treatment. In all, there was present still a frozen shoulder. Three of these patients volunteered the information that following the manipulative procedure their pain had become lessened considerably, and they could move the affected shoulder through a greater arc of motion. Examination of these individuals revealed that the scapulohumeral joint was frozen to a degree comparable with a bony ankylosis. They had even lost the few degrees of motion in the anteroposterior plane which is demonstrable so frequently in patients with frozen shoulders. All motion was in the thoracoscapular joint. The completeness of the lack of glenohumeral motion undoubtedly accounted for the freedom from pain.

CASE REPORTS

CASE M. W. female aged 42 in good health, sustained a fracture of the middle of the shaft of the humerus when she fell down a flight of stairs. The fracture was reduced and the extremity was immobilized in a hanging cast. After 10 weeks the cast was removed at which time the patient noted that she had considerable stiffness in the right shoulder. She volunteered the information that for several weeks prior to removal of the cast she was aware of a dull aching pain in the shoulder which radiated into the neck and into the anterior aspect of the upper arm. Following immobilization the extremity was placed in a sling for a

period of 2 more weeks, during which time she was allowed to exercise her elbow, wrist and fingers, but restoration of function in the shoulder joint was disregarded.

When the patient was seen 12 weeks after injury to the humerus she had a frozen shoulder. The arm was held at the side in a position of internal rotation. There were only about 15° or 20° of motion in the anteroposterior plane and about 10° or 15° abduction at the scapulohumeral joint. All other motion was entirely thoracoscapular. She was started on an intensive program of pendulum exercises which were followed by crawling up-the wall and pulley exercises. Within a period of 8 weeks this patient had regained complete restoration of function in the shoulder joint.

Discussion. This is a typical example of a patient who developed a frozen shoulder following fixation of the extremity in the sling position but because of her general good health and good muscular tonicity succeeded in attaining rapid return of movements by restoration of muscular activity.

Case W S., female aged 42, had a long history of pain and stiffness in the left shoulder which had been initiated approximately 18 months prior to her admission to the hospital by acute and excruciating pain in the anterolateral aspect of the shoulder which radiated into the anterior aspect of the arm and into the posterior cervical region. Pain had been lessened by irradiation but considerable pain and stiffness persisted. Stiffness had become more pronounced within the last 3 months.

Examination 18 months after the initial onset disclosed that the patient had a frozen shoulder except for about 15° of motion in the anteroposterior plane and about 15° of motion in abduction in the scapulohumeral joint. There was marked tenderness throughout the entire anterior and lateral aspects of the joint capsule particularly under the tip of the acromion process. Radiographic examination revealed a large calcareous deposit more or less diffuse in nature involving a large area of the supraspinatus and

the infraspinatus regions of the cuff. At operation a subacute inflammatory process implicating the entire cuff was noted. The biceps tendon was not explored. Large quantities of calcareous material were cutted out of the supraspinatus and the infraspinatus tendons, which also disclosed advanced degenerative changes. There was diffuse infiltration of the material throughout the tendon fibers of the affected cuff. Multiple puncture of the affected portions of the cuff was performed in order to increase the hyperemia and to favor absorption of the remaining calcified material which was not accessible. The subacromial bursa was found thickened, infected and loculated. The thickened walls of the bursa were excised. Following operation the patient was immediately put on a regimen of pendulum exercises followed by crawling up-the-wall and pulley exercises. Restoration of function in the scapulohumeral joint was almost complete at the end of 8 weeks, there was still about 15° residual limitation of motion in abduction, external rotation was possible to 50°, and the patient was completely free of pain.

Discussion. This is an instance in which calcareous tendinitis initiated muscular activity which, in turn, was responsible for a generalized fibrositic process resulting in a frozen shoulder. Removal of the irritant which in this case was calcareous material within the substance of the musculotendinous cuff was sufficient to eliminate pain and initiate muscular activity which in turn restored function to the scapulohumeral joint.

Case B T, female aged 45 became aware of a dull boring ache in the anterolateral aspect of the left shoulder 7 years before the pain gradually increased in intensity was aggravated by motion and relieved by rest. It was worse at night interfering considerably with her rest. She noticed a gradual stiffening of the left shoulder. The range of free painless motion became less and less until she was able to get her hand to her head only with great diffi-

culty. Two years after the onset the patient's arm was manipulated under anesthesia and put on a regimen of physical therapy and exercises. In spite of intensive treatment the patient failed to regain any motion, although her pain did become somewhat less. Thirteen months after the first manipulation, the arm was again manipulated, and another regimen of physical therapy and exercises was instituted similar to that prescribed following the first manipulation. Pain and stiffness persisted. This was followed by a course of irradiation to the shoulder without any relief of symptoms.

Upon examination 7 years after the onset of the symptom complex, the patient presented a very pathetic picture. She was an undernourished, apprehensive, asthenic individual complaining of constant pain in the shoulder; also she had formed the habit of taking large doses of sedatives in order to obtain some rest and sleep. There was considerable atrophy of all the musculature of the shoulder girdle, particularly the spinatus and the deltoid muscles. The arm was held in the sling position. Only 5° or 10° of motion could be demonstrated in the anteroposterior plane; no motion in abduction was demonstrable in the scapulohumeral joint, actively or passively. Pressure over the anterior aspect of the shoulder joint, particularly over the intertubercular sulcus, elicited excruciating tenderness. Rolling of the biceps under the finger also gave rise to considerable pain. Radiographic examination revealed nothing of any significance except that the head rode high in the glenoid cavity and all the bones of the shoulder girdle revealed pronounced calcification. At operation the biceps tendon was found to be firmly adherent within the bicipital groove and also adherent to the undersurface of the joint capsule. Numerous dense adhesions enmeshed the entire tendon within the groove. The subacromial bursa was obliterated completely. There was shortening of all the anterior structures on the anterior aspect of the joint, particularly the subscapularis muscle and the inferior por-

tion of the joint capsule. The entire musculotendinous cuff presented the picture of a low grade inflammatory process.

The biceps tendon was severed at the supraglenoid tubercle and transplanted to the coracoid process. Relief of pain was instantaneous in this patient. Active motion was started on the third day. Following the operation there was progressive increase in the degree of motion. At the end of 14 weeks the patient was able to abduct the arm actively 160°. When last seen the patient still had 10° or 15° residual restriction of abduction and external rotation. However, she was perfectly happy, free of pain, and showed general physical and mental improvement (Fig. 171).

DISCUSSION This case demonstrates that when the pain factor produced by bicipital tenosynovitis (which is always present in a well-established case of frozen shoulder) is removed, restoration of muscular activity can proceed and is followed invariably by increased function at the scapulohumeral joint.

CASE T. R., male, aged 52, sustained severe second-degree burns of the anterior and posterior aspect of the trunk as a result of a gasoline explosion. During the course of treatment both arms were bandaged to his side for a period of 6 weeks. For the following 4 weeks no attention was paid to scapulohumeral motion; the patient was not encouraged to move the extremities. When seen 4 months after his injury the patient complained of severe pain and stiffness in the left shoulder and moderate pain and stiffness in the right shoulder.

Physical examination disclosed a very thin asthenic individual and marked atrophy of the musculature of both shoulder girdles. The left side disclosed a typical frozen shoulder; only a few degrees of motion in the anteroposterior plane and about 10° of abduction were demonstrable in the scapulohumeral joint. Pressure over the intertubercular groove and the biceps tendon elicited exquisite tenderness. Examination of the right shoulder showed from 20° to 30° re-

striction of motion in elevation and 45° of external rotation. Pressure over the intertubercular sulcus and over the biceps tendon evoked tenderness. The above findings were consistent with a bicipital tenosynovitis in the right shoulder. It also became apparent that sooner or later a frozen shoulder would develop. In addition to a frozen shoulder, on the left there was a concomitant bicipital tenosynovitis.

Both shoulders were operated upon in each instance the biceps tendon was transplanted to the coracoid process. Relief of pain was instantaneous and within 8 weeks the patient had complete restoration of function within the right shoulder and within 4 months was able to abduct the left shoulder to 150°, to rotate it externally 45°, and to put his hand in his hip pocket on the affected side without discomfort.

Discussion. This is a typical example of a frozen shoulder following immobilization of the extremity due to extrinsic causes with secondary involvement of the biceps tendon in a fibrositic process affecting all the soft tissues about the shoulder. On the left we had a frozen shoulder in its late stages while on the right side a frozen shoulder in its incipient stage was demonstrable.

BICIPITAL TENOSYNOVITIS

INTRODUCTION

Critical clinical assessment of incidences of painful and stiff shoulders reveals that involvement of the long head of the tendon of the biceps brachii muscle is by far the most common etiologic factor responsible for the symptom complex. This concept is not in agreement with that of many workers interested in shoulder disorders (Codman, Simmonds, etc.) however it has been substantiated many times by visualization of the subacromial area in patients exhibiting the syndrome. Also this investigation forces one to conclude that disorders of the biceps tendon are frequent precipitating causes of frozen shoulder in a great majority of cases.

GENERAL OBSERVATIONS

The clinical material in this investigation fell into two groups. In Group 1 the symptoms were initiated by trauma to the shoulder joint, while in Group 2 the onset was insidious with no definite trauma to the part. Group 1 revealed more men to be afflicted (72.2 per cent) than women (27.8 per cent). Group 2 disclosed a greater prevalence among women (52.3 per cent) than men (47.7 per cent). In this series the right shoulder was involved in 50.5 per cent, the left in 49.5 per cent, and both shoulders in 3.3 per cent. The average age of patients in the first group was 41 years, while in the second group it was 45 years. Regardless of the groups in which they fell no individuals with bicipital tenosynovitis under 30 years of age developed a frozen shoulder. Frozen shoulders were usually encountered in individuals between 40 and 60 years of age. Many of these individuals were below par in general physical fitness. They were apprehensive, emotionally unstable and not a few were victims of chronic debilitating diseases such as pulmonary, cardiovascular and gastro-intestinal diseases. There was a close parallelism between the high incidence of frozen shoulders and the presence of debilitating factors.

BICIPITAL TENOSYNOVITIS ASSOCIATED WITH TRAUMA

General Observations. In bicipital tenosynovitis associated with trauma the usual history is one of pain and stiffness in the region of the shoulder joint following either severe or minor injuries. In this study the severe injuries encountered in the second and the third decades were simple dislocations of the glenohumeral joint, fractures of either tuberosity, surgical neck, fractures and dislocations associated with fractures through the greater tuberosities. In the older cases (after the fourth decade), the most common injuries were fractures of the tuberosities, fractures through the anatomic neck, with or without displacement and fracture

dislocations. The symptom complex not infrequently follows minor injuries, such as a fall on the point of the shoulder or on the flexed elbow joint, forcing the humerus upward. Strenuous exercises, such as tennis or shoveling snow, may initiate the symptoms.

Clinical Features Resulting From Minor Traumata. The outstanding symptom is pain in the anterolateral aspect of the shoulder. This pain is often projected to the region of the insertion of the deltoid muscle and to the belly of the biceps brachii muscle. In severe forms, the pain radiates posteriorly to the region of the scapula and to the posterolateral aspect of the neck. As a rule motion is restricted in all directions, however, a certain range of free voluntary motion is always present. If the arcs of this range are exceeded, the pain may become intense and severe. Abduction and external rotation, and backward flexion and external rotation usually accentuate the pain. Varying degrees of muscle spasm about the shoulder joint are present, all depending upon the severity of the lesion. Such spasm may be demonstrable in the deltoid, the trapezius and the scalenus muscles and at times in the forearm muscles. Pain may be more pronounced at night and interferes with sleep. Upon lying on the affected side, pressure on the point of the shoulder accentuates the pain.

The most constant physical finding, and a very significant one, is exquisite tenderness on pressure over the bicipital groove. Of the several diagnostic tests for bicipital tenosynovitis the one described by Lippmann is the most dependable. Lippmann noted that with the arm actively flexed to a right angle the long biceps tendon can be palpated about 3 inches from the joint. If the tendon at this point is displaced to one side and then suddenly released the patient experiences a sudden twinge of pain in the bicipital groove. It was also observed that with the elbow actively flexed exquisite tenderness was elicited in every case by firmly rolling the long tendon of the biceps under the examiner's thumb. Yergason's

maneuver conveys no information in the majority of the cases.

Roentgenographic examination at this time reveals no information, because the pathologic changes affect the soft tissue structures in the bicipital groove and not the osseous elements of the groove.

In most instances, these acute minor lesions pursue a clinical course which is fairly constant. If the affected extremity is put to rest for several days, the intensity of the symptoms subsides gradually, while the range of motion increases steadily until complete and painless motion is restored.

Occasionally, the acute stage may be followed by a long protracted course of disability of varying intensity. Although there is always a certain range of free and painless motion, if the limits of this motion are exceeded, pain is felt in the anterolateral aspect of the shoulder. This is particularly true of abduction and external rotation and backward flexion and external rotation. The syndrome is accentuated by activity and relieved by rest. Not infrequently pain is referred to the belly of the biceps brachii muscle, to the flexor surface of the forearm and to the scapular region. Pressure over the bicipital groove and rolling the biceps tendon under the examiner's thumb elicits intense pain. Such a cycle may continue for many months. Its intensity varies with the extent of the inflammatory process involving the tendon sheath and the tendon. In one case, such a symptom complex had existed for 3 years.

Occasionally, in individuals past the fourth decade there is a steady increase in the severity of the symptoms, while the arcs of painless motion become progressively smaller until there is little or no scapulohumeral joint motion. The spinati and the deltoid muscles exhibit varying degrees of atrophy. The extremity is held at the side in a position of internal rotation. As a rule a few degrees of free painless motion is always demonstrable in the anteroposterior plane. Clinically the picture now is one of fibrous ankylosis of the scapulohumeral

striction of motion in elevation and 45° of external rotation. Pressure over the intertubercular sulcus and over the biceps tendon evoked tenderness. The above findings were consistent with a bicipital tenosynovitis in the right shoulder. It also became apparent that sooner or later a frozen shoulder would develop. In addition to a frozen shoulder on the left there was a concomitant bicipital tenosynovitis.

Both shoulders were operated upon in each instance the biceps tendon was transplanted to the coracoid process. Relief of pain was instantaneous and within 8 weeks the patient had complete restoration of function within the right shoulder and within 4 months was able to abduct the left shoulder to 150° to rotate it externally 45°, and to put his hand in his hip pocket on the affected side without discomfort.

Discussion. This is a typical example of a frozen shoulder following immobilization of the extremity due to extrinsic causes with secondary involvement of the biceps tendon in a fibrositic process affecting all the soft tissues about the shoulder. On the left we had a frozen shoulder in its late stages while on the right side a frozen shoulder in its incipient stage was demonstrable.

BICIPITAL TENOSYNOVITIS

INTRODUCTION

Critical clinical assessment of incidences of painful and stiff shoulders reveals that involvement of the long head of the tendon of the biceps brachii muscle is by far the most common etiologic factor responsible for the symptom complex. This concept is not in agreement with that of many workers interested in shoulder disorders (Codman, Simmonds, etc.) however it has been substantiated many times by visualization of the subacromial area in patients exhibiting the syndrome. Also this investigation forces one to conclude that disorders of the biceps tendon are frequent precipitating causes of frozen shoulder in a great majority of cases.

GENERAL OBSERVATIONS

The clinical material in this investigation fell into two groups. In Group 1 the symptoms were initiated by trauma to the shoulder joint while in Group 2 the onset was insidious with no definite trauma to the part. Group 1 revealed more men to be afflicted (72.2 per cent) than women (27.8 per cent). Group 2 disclosed a greater prevalence among women (52.3 per cent) than men (47.7 per cent). In this series the right shoulder was involved in 50.5 per cent, the left in 49.5 per cent, and both shoulders in 3.3 per cent. The average age of patients in the first group was 41 years, while in the second group it was 45 years. Regardless of the groups in which they fell, no individuals with bicipital tenosynovitis under 30 years of age developed a frozen shoulder. Frozen shoulders were usually encountered in individuals between 40 and 60 years of age. Many of these individuals were below par in general physical fitness. They were apprehensive, emotionally unstable and not a few were victims of chronic debilitating diseases such as pulmonary, cardiovascular and gastro-intestinal diseases. There was a close parallelism between the high incidence of frozen shoulders and the presence of debilitating factors.

BICIPITAL TENOSYNOVITIS ASSOCIATED WITH TRAUMA

General Observations. In bicipital tenosynovitis associated with trauma the usual history is one of pain and stiffness in the region of the shoulder joint following either severe or minor injuries. In this study the severe injuries encountered in the second and the third decades were simple dislocations of the glenohumeral joint, fractures of either tuberosity, surgical neck, fractures and dislocations associated with fractures through the greater tuberosities. In the older cases (after the fourth decade) the most common injuries were fractures of the tuberosities, fractures through the anatomic neck, with or without displacement and fracture

of the bicipital groove was noted a large hemorrhagic area. It appeared as if blood from a higher level had gravitated distally beneath the synovialis of the floor of the groove. Upon closer inspection it was noted that the uppermost fibers of the subscapularis tendon for a distance of $\frac{1}{4}$ inch, had been torn from their bony attachment on the lesser tuberosity. The torn fibers were frayed, thickened and hemorrhagic. It was apparent that this was the site of bleeding. By splitting the fibrotendinous cuff and capsule, the intracapsular portion of the biceps tendon came into view. It appeared normal in every respect except that the distal portion of the synovialis was greatly injected. The bicipital groove was of normal depth and configuration but it revealed a well formed supratubercular ridge.

The duration of symptoms in the other 6 cases explored ranged from 4 months to 3 years. In Cases C W, P T and W F, the syndrome was rather mild. Motion was normal in all directions, but twinges of pain were felt at the ends of the arc of abduction and external rotation and backward flexion and external rotation.

At operation the tendon sheath in each was found to be thickened, injected and edematous. Numerous long filmy adhesions were disclosed between the tendon and the sheath. In spite of these frail structures, the tendon-sheath gliding mechanism was impaired only slightly. The intracapsular portion of the tendon was free and appeared grossly normal. The extracapsular portion was narrowed. The transition between the two was abrupt. In all a well-defined supratubercular ridge was demonstrable. In W F the inner border of the biceps tendon had made a trough in the uppermost fibers of the subscapularis tendon. No other abnormalities referable to the grooves were noted.

In R M, J S and P S, the syndrome was more severe. Although pain was an outstanding feature, motion in the shoulder joint was restricted and responsible for great disability. In all instances, exploration of the shoulder joint exhibited the sheath to

be reddened, thickened and edematous. The tendon-tendon sheath mechanism was obliterated by well formed vascular adhesions, binding the tendon to the groove. The extracapsular portion of the tendon was thinned, frayed and had lost its sheen.

In Cases R M and J S, the intracapsular portion of the tendon was slightly thickened and edematous, but free. Full passive motion could be demonstrated readily in these cases, in spite of the fact that the tendon was bound to the groove by adhesions which permitted only very limited motion of the tendon in the groove. On the other hand, in Case P S, the intracapsular segment of the tendon was adherent to the inner surface of the capsule in the region of the coracohumeral ligament. Full passive motion in this case was impossible to attain, for intracapsular adhesions acted as mechanical barriers to the excursion of the humeral head on the tendon. Complete motion was attained, however, after the tendon was freed from the inner surface of the capsule.

Four cases aged 38, 47, 58 and 62 with minor injuries to the shoulder joint exhibited a progressive bicipital syndrome which was interrupted by surgical intervention before the phase of complete loss of glenohumeral motion was reached.

The subacromial bursa and the fibrotendinous cuff appeared grossly normal. The biceps tendon was found adherent to the sheath by numerous filmy red adhesions of varying firmness and density. The process extended into the joint binding the distal end of the intracapsular portion of the tendon to the inner surface of the capsule. After the tendon was freed from the sheath and capsule, the range of motion in all shoulders was increased greatly, however, complete motion in abduction and external rotation could not be attained without some force. It was noted for the first time that there had occurred mild contracture of the subscapularis muscle and of the redundant capsule along the inferior aspect of the neck. Abduction and external rotation put these structures on a stretch as more force was

joint or a frozen shoulder. It is of interest to note that in some instances, when all motion is lost in the scapulohumeral joint pain becomes more bearable and it even may disappear. In other instances there is intractable pain.

OPERATIVE FINDINGS A close parallelism exists between the gross pathologic findings and the duration and the severity of the illness. Within a few hours after injury the sheath of the biceps tendon is markedly injected red and edematous thereby constricting the tendon within the sheath. Hemorrhagic areas may be found beneath the synovialis lining the groove actually, tearing of some of the fibers of the subscapularis tendon from the lesser tuberosity on the joint side may be demonstrated. At this stage the process has the appearance of an acute inflammation as yet no definite adhesions are formed. Such findings were exhibited clearly in a shoulder joint which was explored 48 hours after injury (Case C. R.).

Cases with a longer duration exhibit more advanced alterations of the tendon and the tendon sheath. Adhesions varying from fine frail filmy structures to firm dense tough bands of fibrous tissues may be found between the sheath and the tendon. Tendon and sheath may be adherent to the transverse humeral ligament and to the floor and the side of the groove. Occasionally the process may extend into the joint cavity and involve the distal end of the intracapsular portion of the tendon binding it to the inner surface of the capsule in the region of the coracohumeral ligament.

Adhesions within the limits of the groove which obliterate the tendon tendon sheath gliding mechanism or bind the tendon to the groove do not mechanically limit motion. At operation full motion is readily demonstrable without freeing the tendon from the sheath or groove. However if the tendon is adherent to the inner side of the capsule then a definite mechanical barrier exists to free excursion of the humeral head on the biceps tendon. Complete motion is

restored only after the tendon is freed from this attachment. Well formed supratubercular ridges frequently are found diminishing the depth of the groove and often distorting its configuration. In spite of the extensive involvement of the biceps tendon that might be present, changes consistent with those of a frozen shoulder are seldom encountered in young individuals.

CASE REPORTS AND PERTINENT OBSERVATIONS Seven cases all under 40 years of age, with minor injuries were explored. One patient (C. R.) for investigative reasons was explored 48 hours after the initiating trauma.

Case C. R. male aged 23 while serving a tennis ball felt a sudden snap which was associated with excruciating pain in the right shoulder. He was unable to continue the game because all motions of the shoulder joint were painful. During the night his shoulder "throbbled like a toothache." The following day he was seen in the out patient clinic.

Examination disclosed only a few degrees of passive painless forward and backward flexion. Attempted abduction passively or actively was accompanied by severe pain in the anterolateral region of the shoulder joint. Pain was referred also to the anterior aspect of the upper arm. Pressure over the bicipital groove elicited great tenderness. Intense pain was experienced when the tendon of the long head of the biceps muscle was rolled under the examiner's thumb.

At operation the following day 48 hours after the injury the shoulder joint was explored. No pathologic alterations were noted referable to the subacromial bursa. The fibrotendinous cuff was intact and exhibited grossly a normal appearance. Upon splitting the transverse humeral ligament and tendon sheath the biceps tendon bulged into the operative field as if it were released suddenly from great pressure. Its synovial sheath was thickened red and friable. It presented macroscopically the characteristics of an acute inflammatory process. Beneath the synovial lining of the entire length

of the bicipital groove was noted a large hemorrhagic area. It appeared as if blood from a higher level had gravitated distally beneath the synovialis of the floor of the groove. Upon closer inspection it was noted that the uppermost fibers of the subscapularis tendon for a distance of $\frac{1}{4}$ inch, had been torn from their bony attachment on the lesser tuberosity. The torn fibers were frayed, thickened and hemorrhagic. It was apparent that this was the site of bleeding. By splitting the fibrotendinous cuff and capsule, the intracapsular portion of the biceps tendon came into view. It appeared normal in every respect except that the distal portion of the synovialis was greatly injected. The bicipital groove was of normal depth and configuration, but it revealed a well formed supratubercular ridge.

The duration of symptoms in the other 6 cases explored ranged from 4 months to 3 years. In Cases C. W., P. T. and W. F., the syndrome was rather mild. Motion was normal in all directions but twinges of pain were felt at the ends of the arc of abduction and external rotation and backward flexion and external rotation.

At operation the tendon sheath in each was found to be thickened, injected and edematous. Numerous long filmy adhesions were disclosed between the tendon and the sheath. In spite of these frail structures, the tendon sheath gliding mechanism was impaired only slightly. The intracapsular portion of the tendon was free and appeared grossly normal. The extracapsular portion was narrowed. The transition between the two was abrupt. In all a well-defined supratubercular ridge was demonstrable. In W. F. the inner border of the biceps tendon had made a trough in the uppermost fibers of the subscapularis tendon. No other abnormalities referable to the grooves were noted.

In R. M., J. S. and P. S., the syndrome was more severe. Although pain was an outstanding feature, motion in the shoulder joint was restricted and responsible for great disability. In all instances, exploration of the shoulder joint exhibited the sheath to

be reddened, thickened and edematous. The tendon tendon sheath mechanism was obliterated by well formed vascular adhesions, binding the tendon to the groove. The extra capsular portion of the tendon was thinned, frayed and had lost its sheen.

In Cases R. M. and J. S., the intracapsular portion of the tendon was slightly thickened and edematous, but free. Full passive motion could be demonstrated readily in these cases, in spite of the fact that the tendon was bound to the groove by adhesions which permitted only very limited motion of the tendon in the groove. On the other hand, in Case P. S., the intracapsular segment of the tendon was adherent to the inner surface of the capsule in the region of the coracohumeral ligament. Full passive motion in this case was impossible to attain, for intracapsular adhesions acted as mechanical barriers to the excursion of the humeral head on the tendon. Complete motion was attained, however, after the tendon was freed from the inner surface of the capsule.

Four cases aged 38, 47, 58 and 62, with minor injuries to the shoulder joint, exhibited a progressive bicipital syndrome which was interrupted by surgical intervention before the phase of complete loss of glenohumeral motion was reached.

The subacromial bursa and the fibrotendinous cuff appeared grossly normal. The biceps tendon was found adherent to the sheath by numerous filmy red adhesions of varying firmness and density. The process extended into the joint binding the distal end of the intracapsular portion of the tendon to the inner surface of the capsule. After the tendon was freed from the sheath and capsule, the range of motion in all shoulders was increased greatly. However, complete motion in abduction and external rotation could not be attained without some force. It was noted for the first time that there had occurred mild contracture of the subscapularis muscle and of the redundant capsule along the inferior aspect of the neck. Abduction and external rotation put these structures on a stretch as more force was

applied, fine tears were observed in the superficial fibers of the subscapularis tendon and the capsule. These findings are not in agreement with those of Lippmann who maintains that unrestricted motion is attained when the biceps tendon is freed from adhesions within the bicipital sheath and groove.

It was apparent that the afore-mentioned pathologic changes (the bicipital tenosynovitis, contracture of the subscapularis muscle and redundant fibrous capsule) constituted the early phase of a process which, if it progressed, would eventually terminate in extra articular and intra articular fibrous ankylosis of the scapulohumeral joint. Such changes were not noted in younger individuals.

INTERPRETATION OF OPERATIVE FINDINGS AND THEIR CORRELATION WITH THE CLINICAL PICTURE. From the afore-mentioned observations it is obvious that the essential pathologic alteration is an inflammatory process in the sheath of the long biceps tendon and in the tendon itself. The degree and the extent of the inflammatory process governs the character of the bicipital syndrome. Case C R is representative of a very early lesion and it demonstrated that the morbid pathology in cases following severe sudden muscular exertion is one of tearing and stretching of the affected tissues followed by hemorrhage and edema. Pain is due to actual tearing of synovial periosteal and fascial tissues while motion is restricted by the associated reflex muscle spasm about the shoulder joint and by pain. If treated adequately by rest, graduated exercises and physical therapy the inflammatory process retrogresses gradually and complete restoration of function can be anticipated in the majority of the individuals. In the younger age group

In a few instances the illness becomes protracted, the patients develop mild syndromes similar to those of cases C W, P T and W F. Here, the pathologic alterations are not unlike the 'tendovaginitis stenosa' described by Dequervain (1895).

Essentially, the tendon sheath is thickened and it constricts the tendon within, thereby preventing the smooth, free motion of the tendon. Filmy adhesions, present between the sheath and the tendon do not restrict the range of motion; they only add to the mechanical difficulty.

Further progression of the same process results in an obliteration of the tendon tendon sheath gliding mechanism. The tendon and the sheath are bound together by well formed vascular adhesions of varying thickness and toughness or together they may be found adherent to the groove and occasionally to the inner aspect of the capsule in the region of the coracohumeral ligament. Clinically the outstanding features noted in patients with such morbid anatomy are restriction of motion and exquisite tenderness over the bicipital sulcus and the biceps tendon. As previously stated during joint motion adhesions in the groove result in traction on the surrounding periosteal and fascial tissues thereby producing pain. The range of motion is restricted not because of the presence of adhesions in the groove but because of pain and muscle spasm produced when certain arcs of motion are exceeded (Cases R M and J S). However, if the tendon is adherent to the inner surface of the capsule motion is restricted also by a definite mechanical barrier which makes it impossible for the humeral head to glide proximally on the long biceps tendon (Case P S).

Frozen shoulders were not encountered in individuals under 40 years of age regardless of the severity, the extent and the duration of the bicipital tenosynovitis. This observation leads us to assume the presence of some other factor or factors besides tenosynovitis before a frozen shoulder develops. Moreover, the Case of P S demonstrated that a certain range of free motion is possible in the glenohumeral joint even when the biceps tendon is bound to the inner aspect of the capsule. We must admit that the range is greatly restricted by the afore-mentioned mechanical barrier nevertheless.

the clinical picture in no way simulated that of a frozen shoulder.

As previously noted in the majority of instances of acute tenosynovitis, the process gradually subsides with rest to the part, and free full motion is restored. On the other hand, the process may become static for many months with periods of exacerbations, then motion gradually increases, and pain diminishes until normal glenohumeral motion is attained. Knowing the morbid pathology in these cases, the pathologic sequences underlying the clinical events can be reconstructed readily. The tendon tendon sheath mechanism is either restored by gradual dissolution of the binding adhesions or the tendon attains a bony anchorage in the bicipital groove. Obliteration of the tendon tendon sheath gliding mechanism does not bring about freedom of pain. To attain complete and painless motion, the tendon must not have a fibrous attachment to the humerus but a bony one, and there must be no pull on the surrounding tissues. This observation was noted in many cases of this series and will be discussed when the treatment of these lesions is considered.

Clinical Features Resulting From Severe Traumata. In young individuals bicipital tenosynovitis associated with severe traumatic lesions in the region of the shoulder joint is encountered more frequently than hitherto believed. In this series, 8 instances of bicipital tenosynovitis following severe trauma in individuals under 30 years were encountered. It was associated more frequently with depressed fractures of the greater tuberosity than with any other type of lesion. One case revealed on exploration an incomplete tear of the supraspinatus tendon and 3 cases a complete tear. At first the syndrome generally is obscured by symptoms of the more obvious lesion. Involvement of the long head of the biceps tendon and its sheath is manifest by persistence of pain in the anterolateral aspect of the shoulder and by restriction of motion long after the time necessary for healing of bone and soft tissue has elapsed.

Examination discloses, in the acute states, spasm of the short rotator muscles, the trapezius and the scalenus muscles. Depending upon the severity and the duration of the symptom complex, atrophy of the *spinatii* and the deltoid muscles is present in varying degrees. Pressure over the bicipital groove always elicits great tenderness. In every instance, rolling the long tendon of the biceps muscle under the examiner's thumb produces intense pain. Usually, there is observed a certain range of active painless motion. If the arcs of this range are exceeded, severe pain is felt in the region of the bicipital groove. Motions which put the biceps tendon on a stretch as abduction and external rotation or backward flexion and external rotation are accompanied by excruciating twinges of pain.

In some instances, carefully scrutinized roentgenograms give valuable information referable to the bicipital groove. In this series two of the individuals, who had sustained a fracture through the base of the greater tuberosity, exhibited on roentgenographic study considerable new bone formation which encroached upon the outer margin of the bicipital groove (Fig 163). Axial views of the humeral head may reveal spurs on the lesser tuberosity. Although these observations are not diagnostic, they at least provide presumptive evidence of lesions of the biceps tendon when they are associated with a bicipital syndrome.

OPERATIVE FINDINGS. Essentially the pathologic change is an inflammatory process, involving the tendon tendon sheath mechanism. Its severity and extent vary depending in a large measure upon the character of the concomitant lesion.

Fractures in the region of the bicipital groove may change the configuration of the groove thereby impairing the normal gliding apparatus of the biceps tendon (Figs 163 165). Generally, involvement of the tendon and the tendon sheath in these instances surpasses that observed in cases without osseous disruption. The tendon tendon sheath mechanism may be obliterated.

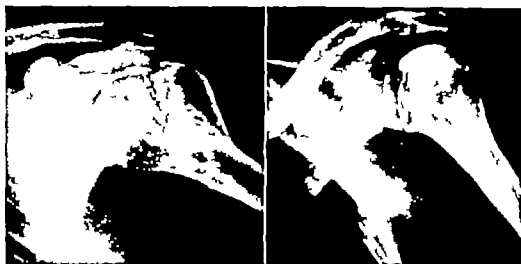


FIG 163 (*Left*) Depressed fracture of the greater tuberosity which healed with pronounced new bone formation changing the configuration of the bicipital groove. At operation, in addition to the above osseous changes the biceps tendon was found adherent to the distorted groove by dense firm adhesions (case L. P.)

FIG 164 (*Right*) Unusual depressed fracture of a portion of the head of the humerus resulting from shock therapy in a female, aged 52 years. In spite of immediate mobilization and physical therapy, a frozen shoulder developed. At operation the intertubercular sulcus was found to be narrowed and irregular and the constricted biceps tendon was enmeshed in filmy vascular adhesions.

ated by dense firm vascular adhesions which bind the entire mass to the groove. The tendon is frayed, shredded and thinned; only a thin fibrous band in the groove may represent the remains of the tendon. This process extends into the capsule and may bind the distal end of the intracapsular portion of the tendon to the inner surface of the capsule. As a rule the groove is shallow and narrow. Bony spurs are frequent findings on the inner and the outer boundaries and occasionally in the floor of the groove. With depressed fractures of the greater tuberosity new bone formation along the upper and outer margin of the bicipital sulcus is a common finding. These bony excrescences not only decrease the depth and the width of the groove but actually dig into the substance of the tendon during joint motion (Fig. 166).

CASE REPORTS AND PERTINENT OBSERVATIONS. Four individuals with depressed fractures of the greater tuberosity were explored. In all the tendon was found adherent to the sheath and the groove and to

the inner surface of the capsule in the area of the coracohumeral ligament. At operation full motion could not be demonstrated after the biceps tendon was freed in the groove; it could be accomplished only after the tendon was released from the inner surface of the capsule.

In spite of the extensive involvement of the biceps tendon and the tendon sheath, frozen shoulders were not encountered in this younger age group.

Case J. C. male, aged 30, wrenched his shoulder while wrestling. Pain was localized in the anterolateral aspect of the shoulder and radiated into the forearm. It was more intense at night and any activity aggravated his symptoms. He was treated by physical therapy for a year without improvement. Four years after the injury he was seen in an Army hospital complaining of constant pain in the anterolateral aspect of the shoulder joint. The pain radiated anteriorly to the belly of the biceps brachii muscles and into the posterior region of the scapula.



FIG. 165 Other types of osseous lesions implicating the bicipital groove and the bicipital tendon tendon sheath mechanism, which were responsible for the bicipital syndrome in this series. In both instances, implication of the biceps tendon was confirmed at operation. Both developed frozen shoulders.

Examination disclosed moderate atrophy of the spinatus muscles. There were a few degrees of painless active voluntary motion in the anteroposterior plane. Any attempts of abduction passively or actively accentuated the pain. Pressure over the greater tuberosity and over the bicipital groove elicited exquisite tenderness. Roentgenographic examinations exhibited no pathologic alterations. A tentative diagnosis of rupture of the supraspinatus tendon was made, and an exploratory operation of the shoulder was recommended.

At operation, it was noted that the walls of the subacromial bursa were thickened and injected. The floor of the bursa was red and had lost its luster. Although no complete rupture of the fibrotendinous cuff in the region of the insertion of supraspinatus tendon was found, the cuff in this region was very thin, permitting the underlying bone of the greater tuberosity to be readily palpable. The thin, frayed portion of the cuff was resected, thereby bringing the distal end of the intracapsular portion of the biceps tendon into view. Inspection of the

tendon disclosed it to be free in the joint; however, it was injected, thickened and had lost its sheen. Fine adhesions were found between the under surface of the tendon and the floor of the upper end of the intertubercular groove. These findings were consistent with a tenosynovitis, yet their significance at this time was not fully appreciated. No exploration of the tendon within the groove was carried out. In the belief that thinning, fraying and shredding of the supraspinatus tendon were the lesions responsible for the symptom complex, the impaired portion of the tendon was excised. The healthy end of the supraspinatus tendon was sutured, without difficulty, to the greater tuberosity in the conventional manner.

In spite of intensive physical therapy, no demonstrable improvement in the patient's symptoms or range of motion was noted 8 months after operation. Pressure over the bicipital groove at the level of the tuberosities elicited great tenderness. Rolling the biceps tendon under the examiner's thumb produced severe pain. Attempts at abduc-

tion and dorsal flexion and external rotation accentuated the pain. A diagnosis of bicipital tenosynovitis was made. At operation the bursal walls were found to be greatly thickened; the bursa was loculated. The

About an inch of the distal portion of the intracapsular tendon was firmly adherent to the inner surface of the capsule in the region of the coracohumeral ligament; the tendon could be freed only by sharp dissec-



FIG. 166 (Left) Specimen obtained from a cadaver in which there is evidence of a depressed fracture of the anatomic neck and the head of the humerus. The upper portion of the intertubercular sulcus has been completely obliterated and is filled in with numerous bony excrescences. The inferior surface of the corresponding biceps tendon exhibited advanced degenerative changes. (Right) Specimen obtained from a cadaver which had sustained a fracture through the surgical neck. Note the complete obliteration of the bicipital groove and marked irregular new bone formation. In this specimen the extracapsular portion of the biceps tendon was found attached to this bony mass. The intracapsular portion was absent.

fibrotendinous cuff was intact; the only evidence of repair was the presence of several black sutures which were removed. Upon splitting the transverse humeral ligament the biceps tendon was found to be firmly attached to the roof of the ligament and to the floor of the groove by firm dense adhesions. The tendon was narrowed, injected and roughened. By splitting the musculotendinous cuff through the outer fibers of the coracohumeral ligament as far as the apex of the glenoid cavity the intracapsular portion of the tendon was plainly visualized.

Free full passive motion was possible after it was released from the inner surface of the capsule. The remainder of the tendon appeared thickened, red and edematous. The tendon was severed close to the supra-glenoid tubercle and was withdrawn from the joint cavity. The edges of the fibrous capsule and cuff and the transverse humeral ligament were approximated by interrupted fine silk sutures. About an inch of the biceps tendon was removed for histologic study and the end was then fixed to the tip of the coracoid process. Relief of pain in this indi-

vidual was almost dramatic. Two weeks after operation he was able to abduct the arm actively to 110° without pain, 3 weeks later abduction was possible to 160° , and 10 weeks later he had complete and painless motion in the shoulder joint.

Three other cases (W P, S T and R V) disclosed a complete tear of the supraspinatus tendon associated with a bicipital tenosynovitis. In width, the tears measured approximately $\frac{1}{2}$ in, $\frac{3}{8}$ in and 1 in respectively. In each instance, the biceps tendon was extensively involved, the tendon sheath mechanism was obliterated by numerous adhesions, and the tendon was adherent to the under surface of the capsule. As in the case of J C, full range of motion was possible only after the tendon had been released from the inner surface of the capsule in the region of the coracohumeral ligament. No attempt was made to repair the defects in the supraspinatus area of the fibrotendinous cuffs. In all 3 instances the biceps tendon was transplanted to the coracoid process. All made an uneventful recovery and there was complete restoration of painless motion in from 8 to 10 weeks.

Study of these cases revealed many pertinent observations. It became apparent that lesions of the biceps tendon may be associated with tears of the musculotendinous cuff. Although Codman was of the opinion that partial or incomplete tears of the supraspinatus tendon (tears on the joint side of the supraspinatus tendon) were the most common causative lesions responsible for painful and stiff shoulders, these cases demonstrated that involvement of the biceps tendon may play an even more important role. From this study it is reasonable to assume that tears of the supraspinatus tendon are compatible with good shoulder function provided that the other rotators are intact and capable of fixing and depressing the humeral head in the glenoid cavity during abduction and elevation of the arm. These cases also demonstrated emphatically that mechanical impairment of motion is due to adhesions between the biceps tendon

and the inner surface of the capsule. Furthermore, following Hitchcock and Bechtol, it was noted that by removing the entire tendon from the joint cavity and transplanting it to some other point, the patients were relieved immediately of pain, and free painless, shoulder motion was regained rapidly.

Case L P., a male, aged 37, sustained an injury to the right shoulder when he fell out of a weapon carrier. His arm was placed in a plaster shoulder spica for 12 weeks. After a year of physical therapy, he still complained of pain and stiffness in the right shoulder. When the patient was seen 2 years after the injury, the pain was constant, and all motions of the shoulder joint accentuated the pain. It was most severe at night, especially if he rested on the affected shoulder. He localized the pain at the insertion of the deltoid muscle and in the anterior aspect of the upper arm. Tingling and numbness was occasionally felt in the forearm and the hand along the course of the ulnar nerve.

Examination disclosed advanced atrophy of the spinatii muscles and moderate atrophy of the deltoid muscle. Although he complained of pain, he was able to abduct the arm voluntarily to 60° . Beyond this arc, he experienced severe twinges of pain causing him to drop the arm to the side. Motion in the anteroposterior plane was free if he did not exceed 45° forward or dorsal flexion. With the arm at the side, 50° of active and passive painless external rotation was present. Pressure over the bicipital groove evoked great tenderness, a similar response was obtained when the biceps tendon was rolled firmly under the examiner's thumb. Pain was elicited at the root of the neck when pressure was made in the supraclavicular space over the scalenus anticus muscle. This maneuver accentuated the pain in the ulnar aspect of the forearm.

Roentgenographic study disclosed an old healed depressed fracture of the greater tuberosity (Fig 163). Considerable irregularity was demonstrable over the superior surface of the tuberosity and along the



FIG 167 Intracapsular portion and a small segment of the extracapsular portion of the biceps tendon removed from the shoulder (case L. P. cf Fig 163). The tendon is attenuated in its middle and thickened at either end; the constricted portion was firmly bound down in a narrowed irregular intertubercular sulcus.

lateral margin of the intertubercular groove.

A diagnosis of bicipital tenosynovitis with an associated scalenus anticus syndrome was made.

Exploration was carried out through an anterior muscle-splitting incision. The subacromial bursa was found to be obliterated completely; the subdeltoid fascia was adherent to the greater tuberosity and to the outer surface of the musculotendinous cuff. Considerable irregular new bone formation was noted over the superior surface and along the lateral margin of the intertubercular groove. Bony spurs projected from the lateral wall of the groove. The configuration of the groove was distorted. By splitting the transverse humeral ligament the fibrous capsule and the coracohumeral ligament of the extracapsular and intracapsular portions of the biceps tendon were visualized clearly. The tendon was found to be bound firmly by dense adhesions to the floor and the roof of the intertubercular groove. Moreover that portion lying in the groove was reduced in thickness and presented no trace of a synovial lining. Beginning at the level of the tuberosities and extending upward into the joint for 2 cm, the tendon was adherent to the undersurface of the capsule in the region of the coracohumeral ligament. Sharp dissection was necessary to free the tendon from its bed of fibrous tissue (Fig 167).

Gross inspection of the musculotendinous cuff revealed no pathologic alterations. Before freeing the tendon the arm could

be abducted passively 90°. A definite mechanical restriction was felt if the patient attempted to exceed this arc. It was noted that as the arm came up to 90° the hypertrophied greater tuberosity abutted against the coraco-acromial ligament. No increase in range of motion was noted when the tendon was freed from adhesions in the groove but when it was released from the inner surface of the joint capsule the arm could be abducted fully; however, the coraco-acromial ligament became taut as the greater tuberosity passed under it. It was apparent that in this instance the coraco-acromial ligament was also a barrier to free abduction of the arm.

The biceps tendon was severed close to the supraglenoid tubercle and withdrawn from the joint. It was then anchored to the tip of the coracoid process. The excess new bone over the greater tuberosity was removed with an osteotome. The coraco-acromial ligament was divided close to the inner border of the acromion process and the margins of the coracohumeral ligament and the transverse humeral ligament were approximated with fine silk sutures.

Motion was started on the third postoperative day. There was an immediate and progressive increase in the range of painless motion so that at the end of the third week abduction was possible to 130°. Eight weeks after operation motion was complete and painless in all directions. All symptoms indicative of a scalenus anticus syndrome disappeared.

This case emphasizes the possibility of the existence of a scalenus anticus syndrome with lesions of the biceps tendon. Although the syndrome is as a rule a secondary phenomenon it may assume such prominence that the primary lesion may be obscured. Again it was noted that adhesions in the groove are only of significance in that they make tension upon the surrounding periosteal and fascial tissues thus producing pain and muscle spasm which restrict motion; they are not mechanical barriers to free motion. This is substantiated by the

clinical observation that once the biceps tendon has attained a bony anchorage to the shaft of the humerus, either by surgical intervention or spontaneously, pain disappears, and restoration of function is assured. On the other hand, as previously mentioned, adhesions binding the tendon to the inner side of the capsule in the region of the coracohumeral ligament not only cause pain and muscle spasm but also restrict the excursion of the humeral head on the biceps tendon during shoulder motion.

Three other cases (T. C., V. M. and B. D.) with depressed fractures of the greater tuberosities and an associated bicipital syndrome were explored. Pathologic findings, management and response were similar to those recorded in the case of L. P. It was significant to note that, regardless of the extensive involvement of the biceps tendon in this younger age group, no instance of frozen shoulder was encountered.

Severe injuries about the shoulder joint are not uncommon in individuals past the third decade. If adequately treated, it is common knowledge that such injuries are compatible with good shoulder function. However, regardless of the efficacy of treatment, there are many instances in which pain and stiffness of the shoulder joint persist. This is especially true of lesions in the region of the bicipital groove, such as fractures through the tuberosities, depressed or comminuted fractures of the humeral head and fractures through the anatomic neck. Involvement of the biceps tendon may be primary or secondary to changes in the configuration of the bicipital groove. The illness may pursue several different courses; it may be similar to that recorded in the cases of J. C. and L. P. after many weeks it may retrogress gradually while motion steadily increases until full free motion is attained or the syndrome may progress until there is complete loss of scapulohumeral motion.

It was noted in this study that severe lesions did not necessarily predispose one to the development of a frozen shoulder. On the other hand, the general physical fitness

of the patient appeared to be an important deciding factor. In this series, 6 individuals (4 females and 2 males, all over 50 years of age), following fractures of the humeral head, developed a bicipital syndrome which terminated in a frozen shoulder. All 6 were below par physically, also, they were apprehensive and emotionally unstable.

Any lesion of the upper end of the humerus which disrupts the normal configuration of the bicipital groove may initiate a bicipital tenosynovitis. In this age period there were 3 cases with a depressed fracture of the greater tuberosity, one with a fracture through the base of the lesser tuberosity with inward displacement of the fragment, 4 with fracture of the greater tuberosity and dislocation of the humeral head, 1 with fracture through the anatomic neck, and 1 with a depressed fracture of the humeral head. All these cases were explored, and involvement of the long tendon of the biceps was demonstrable. As previously stated, 6 of these developed frozen shoulders. In 3 individuals (Figs 164 and 165) aged 52, 57 and 49 respectively, the bicipital syndrome developed and terminated in a frozen shoulder, regardless of the fact that every precaution was taken to mobilize the shoulder joint as early as possible.

Codman was of the opinion that depressed fractures of the greater tuberosity always were associated with traumatic tendinitis of the rotator tendons, which in turn was responsible for pain, stiffness and protracted disability of the shoulder. He completely overlooked the long head of the biceps tendon as a causative agent in these instances. He failed to appreciate the significance of a distorted irregular bicipital groove in the production of bicipital tenosynovitis, which may initiate processes responsible for frozen shoulders in the older individuals.

BICIPITAL TENOSYNOVITIS WITHOUT TRAUMA

Bicipital syndromes initiated by or following trauma have been considered thus far in this presentation. There are, however

a far greater number of individuals with clinical pictures identical with those described without definite histories of injury. These individuals, like those previously discussed, fall into two groups: a younger age group under 30 years of age, and an older age group over 30 years of age. The distinguishing feature between the two is that frozen shoulders are more prone to develop in the latter than in the former group.

Younger Age Group. As a rule the onset is an insidious one. A mild ache is noted in the anterolateral aspect of the arm and twinges of pain are felt at the limits of normal arcs of motion. Not infrequently pain is projected to the anterior aspect of the upper arm and to the insertion of the deltoid muscle. Pain is accentuated by increased activity and is relieved by rest. The arcs of abduction and external rotation and backward flexion and external rotation may be decreased. Increased pain at night is a common complaint. The most significant physical findings are tenderness revealed on pressure over the bicipital groove and also on rolling the long head of the biceps tendon under the examiner's thumb.

The clinical course of the illness is very unpredictable; it may remain in a static state, neither progressing or retrogressing for many months; on the other hand it may disappear completely for varying periods of time, only to reappear again. In one individual in this series the duration of the syndrome was 11 years.

Three individuals, when the arm was abducted and rotated externally, felt a definite snap in the shoulder which was accompanied by lancinating pain in the bicipital area. They could produce the snap voluntarily. In one patient both shoulders were so afflicted.

In this investigation frozen shoulders were not encountered in the younger age group. One patient aged 31 developed a frozen shoulder. However, the duration of her illness was 11 years, during which time she had a rather free range of scapulohumeral motion; pain was experienced only at the limits of this range.

OPERATIVE FINDINGS. Seven patients in this younger age group were explored. No significant findings were noted referable to the subacromial bursae and musculotendinous cuffs. In all, the biceps tendon and its sheath disclosed alterations similar to those in the cases of C. W., P. T. and W. F., (p. 153) which changes were consistent with tenosynovitis. In two shoulders the groove was shallow and a pronounced supratubercular ridge was present. Three patients with snapping shoulders revealed some noteworthy findings. In one patient there was present an unusually high supratubercular ridge and a shallow groove. The biceps tendon was displaced to the inner side of the ridge and lay on the lesser tuberosity in a fascial sling made up of the uppermost fibers of the subscapularis tendon. Both biceps tendon and tendon sheath were covered by frail, red, filmy adhesions; the sheath was reddened, thickened and edematous. Although the other two patients with snapping shoulders failed to demonstrate such a prominent supratubercular ridge, a definite ridge was present, and the bicipital grooves at the level of the tuberosities were shallow. In one of these the biceps tendon was found displaced completely out of the groove and had made for itself a bed in a fascial sling on the inner side of the lesser tuberosity.

COMMENT. Analysis of this group leads one to conclude that certain configurations of the bicipital sulcus may be the underlying factors responsible for tenosynovitis in some individuals. The frequency with which a supratubercular ridge, a shallow bicipital groove or both occurred in the shoulders explored in this study is significant. These findings confirm those of Hitchcock and Bechtol. However, one must admit that other factors such as occupation, overuse or even physiologic wear and tear may initiate such a syndrome in the presence of a normal groove, for as has been shown, the long biceps tendon works at all times at a great mechanical disadvantage.

Older Age Group. In this group the onset and the course of the illness is similar

to that recorded for the younger age group, except that older individuals are more prone to develop frozen shoulders. This is especially true in individuals who are below par physically, or when the lesion is associated with some debilitating disease. It was noted in this investigation that regardless of what other factors are responsible for frozen shoulders, increasing age plays a definite role. Once a frozen shoulder is developed it pursues a clinical course similar to that described in older individuals, with frozen shoulders following minor or severe injuries about the shoulder joint.

That frozen shoulder is not ushered in always by a bicipital tenosynovitis is illustrated by the following cases.

Three women (H. B., C. R. and V. T., aged 47, 53 and 54, respectively) developed frozen shoulders subsequent to fractures of the distal end of the radius and the ulna. Following reduction of the fractures the arms were immobilized in plaster casts and supported in slings. Maintenance of shoulder motion was disregarded during the period of immobilization of the fractures. Prior to the injuries all these individuals stated that there were no symptoms or signs which were indicative of pathologic disorders in their shoulders. All had complete and painless motion. From 3 to 6 weeks after the injuries, they became aware of stiffness and a dull ache in the shoulder on the affected side. These symptoms progressed steadily. When the patients were examined (H. B. 8 weeks, C. R. 11 weeks and V. T., 16 weeks respectively) after their injuries all disclosed typical frozen shoulders. Another individual T. W. aged 54, sustained an injury to the dorsum of the right hand which necessitated a skin graft. He too, had no symptoms referable to his right shoulder prior to the time this operative procedure was undertaken. In order to swing a full thickness pedicle skin graft from the abdomen to the dorsum of the right hand the upper extremity had to be placed in a position of complete adduction and internal rotation. This position was maintained by plaster swathes for 4 weeks.

During this period he was conscious of a constant, deep-seated pain in the right shoulder, the pain being more severe at night. At the time the graft was released from the abdomen, it was noted that there existed a frozen shoulder. Only a few degrees of scapulohumeral motion were demonstrable in the anteroposterior plane.

The affected shoulders of three of these patients were explored. Varying degrees of inflammation were disclosed in the subacromial bursae. Essentially, the changes noted were increased congestion and thickening of the roof and the floor of the bursa. One bursal sac was loculated by several firm dense adhesions. In all, the musculotendinous cuff and inferior portion of the capsule were adherent to the humeral head. Any attempt at internal or external rotation or abduction put these structures on a stretch. If the forces of abduction and external rotation were continued, tears in the subscapularis tendon and in the lower subscapularis muscle fibers were plainly discernible.

All three bicipital tendon sheaths were thickened, edematous and injected. Numerous vell like, red adhesions were demonstrable between the tendon and the tendon sheath and the roof and the floor of the groove. The process extended into the joint cavity involving the distal end of the intracapsular portion of the biceps tendon and the adjacent surface of the capsule.

Grossly, the findings were that of an inflammatory process involving the capsule, the musculotendinous cuff, the synovial membrane, the biceps tendon and, to a lesser degree, the subacromial bursa. In the light of these observations, it is reasonable to conclude that the involvement of the biceps tendon and its sheath was secondary to a generalized inflammatory reaction affecting all structures in this region. However, it becomes obvious that the obliteration of the tendon-tendon-sheath mechanism may play a major role in retarding recovery. Therefore, the illness must now pursue a clinical course similar to that described in patients with frozen shoulders which were initiated

by a bicipital tenosynovitis of traumatic or nontraumatic origin

Treatment. From this study it is apparent that bicipital tenosynovitis should be treated conservatively regardless of the causative agents in the early stages. Rest to the part and restriction of motion within the painless arcs usually suffice to terminate the syndrome. Surgical intervention is justified only in those instances showing a steady progression of the intensity of the illness

which fails to respond to conservative measures, also, it is justified in individuals in whom there are repeated recurrences of the symptom complex which results in much discomfort and protracted disability. The operation preferred is transplantation of the biceps tendon to the coracoid process.

As the result of this investigation, one is forced to concede that our present-day concept of the management of severe fractures about the humeral head and tears of the

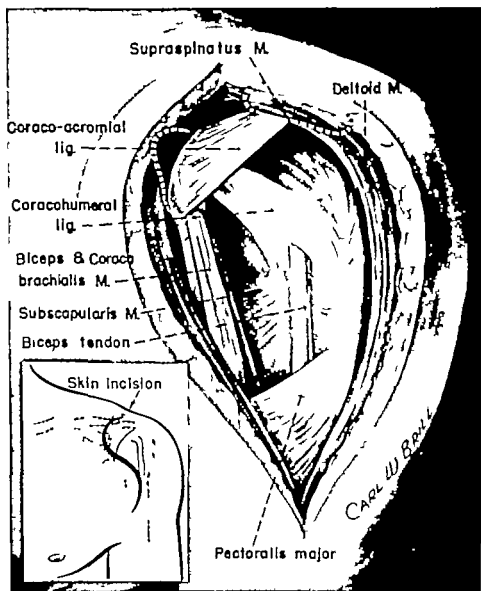


FIG 168 Exposure of anterior aspect of the shoulder by the S-shaped skin incision of Hitchcock and Bechtol as shown in insert. The deeper structures are exposed by developing the deltopectoral cleft and retracting the deltoid muscle laterally and the pectoralis major muscle medially. Approximately 1 centimeter of the medial border of the deltoid muscle together with the cephalic vein are displaced medially with the pectoralis major muscle.

musculotendinous cuff, involving chiefly the supraspinatus tendon requires some revision. Considering the frequency of bicipital tenosynovitis following humeral head fractures, one should anticipate this sequela. In instances which demand surgical intervention, the bicipital area should be investigated, and, if there is evidence of involvement of the tendon tendon sheath

mechanism, the tendon should be transplanted to the coracoid process as a primary measure. This is especially true of the following types of cases: (1) cases with fracture dislocations of the humeral head in which the biceps tendon has been dislocated from the upper portion of the groove; (2) cases of fractures of the greater tuberosity, with retraction of the fragments under

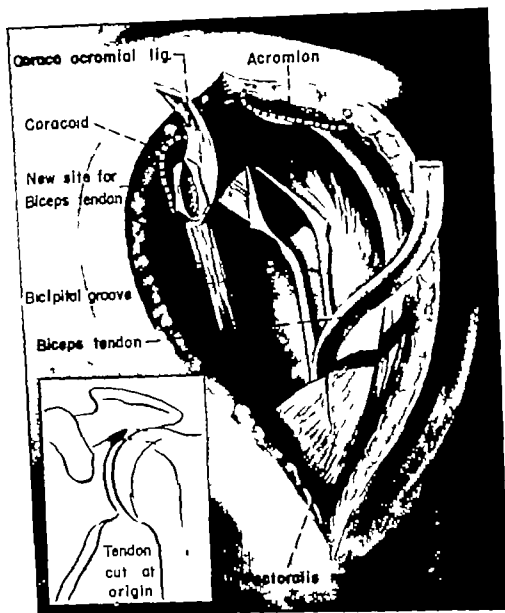


FIG 169 The intertubercular groove is opened by a longitudinal incision through the transverse humeral ligament the incision is extended through the fibers of the coracohumeral ligament in the interval between the subscapularis and the supraspinatus muscles next, the biceps tendon is severed at the superior glenoid rim and withdrawn from the joint cavity. The coraco-acromial ligament is divided close to its insertion into the acromion also the tendinous fibers of the insertion of the pectoralis major are divided for 2 to 3 centimeters. A bony trough is cut out of the coracoid process.

the acromion in which the fracture line traverses the bicipital sulcus and (3) cases of depressed fractures of the greater tuberosity in which the biceps tendon is compressed by displaced fragments of bone. In 8 individuals who had lesions similar to those just enumerated the biceps tendon was transplanted to the coracoid process; in no instance did a bicipital tenosynovitis develop. In 6 others in whom the tendon was not transplanted a typical bicipital syndrome developed. Two of the 6 terminated in a frozen shoulder. This investiga-

tion further emphasizes the clinical observation that rupture of the supraspinatus tendon is compatible with good painless shoulder function and that if pain and limitation of motion are present the most likely underlying etiologic factor is a bicipital tenosynovitis.

Surgical intervention is also justifiable in individuals with a bicipital syndrome in whom a frozen shoulder appears inevitable.

OPERATIVE TECHNIC The anterior portion of the deltoid muscle is exposed through an S-shaped skin incision. After splitting the

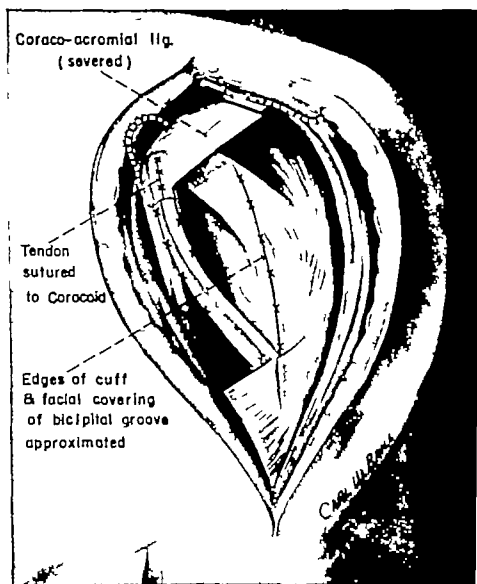


FIG. 170 The biceps tendon is sutured in the trough in the coracoid process and to the tendon of the short head of the biceps brachii muscle. The incision in the coracobrachial and transverse humeral ligaments is closed by interrupted sutures.

deltoid muscle fibers the musculotendinous cuff and the subacromial bursa are carefully inspected. By rotating the humerus inward and outward, the entire tendinous portion of the rotator cuff can be visualized. The transverse humeral ligament and the capsule bridging the intertubercular area are then incised longitudinally, thereby exposing the extracapsular portion of the biceps tendon. This incision is extended proximally through the outer fibers of the coracohumeral ligament for a distance of 2 inches. Such an exposure provides adequate visibility of the intracapsular portion of the biceps tendon. Next, the biceps tendon is severed close to its site of origin from the apex of the glenoid cavity and withdrawn from the joint cavity.

A vertical incision $\frac{1}{2}$ inch long is made over the tip of the coracoid process through the fibers of the coracobrachialis and the short head of the biceps brachii muscles which arise from this point. The coracoid process is exposed subperiosteally and, with a small gauge, a vertical trough is made in its substance. The biceps tendon is sutured in the bony trough and covered with periosteum and the tendinous fibers of the cor-

acobrachialis and the short head of the biceps muscles. About 2 inches of the proximal end of the biceps tendon is sutured to the combined tendon of the coracobrachialis and the short head of the biceps muscle with medium silk sutures. Fine silk sutures are used to approximate the edges of the incised fibrous capsule and the transverse humeral ligament. The wound is then closed in the usual manner. The arm is not forced through its normal range of movements at the glenohumeral joint.

No external fixation is employed other than a sling. Pendulum motion is started the next day and is performed a prescribed number of times every hour on the hour by the clock. The sling is discarded after 5 days. The range of motion is increased progressively, but it is kept always within the tolerance of the patient. Optimum return of function is gradually restored, the time varies from 3 to 4 months; however, the patients are now free of pain.

In all instances in which this procedure was carried out the patients were dramatically relieved of their old pain and restoration of motion was initiated promptly. This

FIG. 171 Range of abduction and external rotation of the affected extremity (left) 4 months following transplantation of the biceps tendon to the coracoid process (case B. T.)



was true not only of cases with uncomplicated tenosynovitis, but also of cases with frozen shoulders

CORACO-ACROMIAL LIGAMENT

Frequently this structure acts as a definite mechanical barrier to the humeral head when the extremity is abducted. Division of the ligament allows the head to pass beneath the coraco-acromial arch with considerable freedom. This procedure is found to be especially useful in frozen shoulders in which the head has risen high in the glenoid cavity and abuts against the coraco-acromial ligament. At this point a review of the anatomy of the coraco-acromial ligament is indicated. It is a triangular structure whose apex inserts into the inner side of the acromion while its broad base inserts into the outer border of the coracoid process. Its inner and outer portions are strong; its center is weak. In fact in some instances the central area consists of fine areolar tissue traversed by weak fibrous strands. This anatomic arrangement demands that if the ligament is divided it should be severed close to its insertion into the acromion in order to release all its fibers (Fig 13)

CORACOHUMERAL LIGAMENT

As previously stated, the coracohumeral ligament with the arm at the side and rotated internally is in a relaxed and shortened state. In frozen shoulders this structure is similarly involved as are all the soft tissue elements comprising the rotator muscles and the musculotendinous cuff. It is converted into a strong thickened shortened fibrous band extending from the horizontal limb of the coracoid process to the fibrous capsule as far as both tuberosities. In this form the ligament acts as a powerful checkrein to external rotation and abduction. Therefore, it becomes apparent that division of this structure will promote early restoration of glenohumeral motion. In all operative procedures on frozen shoulders this ligament has been severed along its line of insertion into the coracoid process.

The operative technic described was adopted because of the unsatisfactory results obtained with other procedures. Lippmann's method of fixation of the biceps tendon was performed in the first 6 cases of bicipital tenosynovitis, 2 of which exhibited also frozen shoulders. In all the convalescence was a protracted one. In spite of intensive physical therapy restoration of motion was slow and painful. Although these individuals made a good recovery eventually, the average time to obtain this result was 5½ months. This method was discarded, and one similar to that described by Hitchcock and Bechtol was performed on the next 6 cases. The results in this series showed no improvement over the previous group. Finally the surgical procedure herein described was evolved and has given satisfactory results in a shorter period of time.

SUMMARY

- 1 Bicipital tenosynovitis is not an uncommon lesion causing pain and stiffness in region of the shoulder joint. It may be associated with minor or severe injuries about the shoulder or it may have no relation to trauma.

- 2 Because of morphologic changes in the humerus and the scapula and because of the demands made on a prehensile extremity the biceps tendon functions at a great mechanical disadvantage. These factors render the tendon vulnerable to repeated trauma during normal joint motion.

- 3 Grossly macroscopic degenerative alterations were noted in the musculotendinous cuff, the capsule and the synovials in the fifth decennium. Microscopic changes were discernible in the third decade. These lesions increased in gradient in each successive decade pointing to the parallelism between degenerative lesions and senescence.

- 4 Regardless of the cause no instance of bicipital tenosynovitis under 30 years of age terminated in a frozen shoulder. Frozen shoulders were encountered most frequently after 40 years of age.

- 5 Any agent which restricts scapulohu-

meral motion may initiate the processes responsible for frozen shoulders, provided that there are present degenerative changes in the soft tissue elements of the scapulohumeral joint. Bicipital tenosynovitis is the most common etiologic factor.

6 Essentially, these processes comprise slowing of the circulation, venous and lymphatic stasis, accumulation of serofibrinous exudates, and formation of adhesions in and about all the soft tissue structures of the scapulohumeral joint, including the biceps tendon tendon sheath mechanism. In the younger age groups these processes do not progress to the degree which result in frozen shoulders, while after 40 years of age, in which degenerative lesions are readily demonstrable, frozen shoulders are not infrequent sequels.

7 Adhesions involving the tendon tendon sheath mechanism afford a mechanical barrier to excursion of the humeral head on the biceps tendon only when they bind the tendon to the inner surface of the capsule.

8 Bicipital tenosynovitis is a frequent concomitant lesion with fractures of the upper end of the humerus and also associated frequently with lesions of the musculotendinous cuff without fractures.

9 Bicipital tenosynovitis associated with supraspinatus tears may be the responsible lesion for the symptoms. Supraspinatus lesions are compatible with painless and complete shoulder motion. These observations are not in agreement with Codman, who was of the opinion that supraspinatus lesions (partial or complete) were the most common factors responsible for painful and stiff shoulders.

10 An operative procedure is offered for treatment of bicipital tenosynovitis and frozen shoulder.

RUPTURE OF THE BICEPS TENDON

PROXIMAL TENDON RUPTURES

General Considerations. Rupture through a normal tendon rarely occurs. A

few instances have been recorded, however, in which rupture of the biceps tendon followed violent unguarded exertion in young athletes. As a rule, the lesion is encountered in those past middle life and it occurs in tendons exhibiting varying degrees of degeneration. As pointed out in the second part of the discussion of degenerative lesions of the shoulder joint, macroscopic manifestations of degeneration in the tendon are common findings in normal shoulders. They increase in severity with each subsequent decade and result from aging and attrition. Hypertrophic bony alterations in the articular cartilage of the head of the humerus, the tuberosities and the intertubercular groove are partially responsible for such attritional changes as shredding, fraying and, finally, partial or complete rupture of the tendon fibers.

Spontaneous rupture is not an infrequent occurrence in shoulders possessing advanced degenerative lesions in the cuff and the head of the humerus. In some shoulders with marked degenerative lesions before complete severance of the tendon occurs, the extracapsular portion of the tendon attains a bony attachment on the shaft of the humerus below the lesser tuberosity. Therefore it is apparent that forceful contraction of the biceps brachii muscle may produce a partial or complete rupture of such degenerative tendons.

Meyer pointed out that occupation may play a major role in accentuating the degenerative lesions associated with aging, especially when the arms are used in a position of external rotation and abduction. This forces the tendon firmly against the lesser tuberosity, favors subluxation of the tendon and produces frictional forces, which cause fraying, shredding and tearing of tendon fibers. Excessive fatigue superimposed upon degenerative and attritional changes may result in rupture of the tendon. Gil creest reported that 83 per cent of the ruptures of the tendon are caused by indirect trauma (contraction of the biceps muscle). Direct trauma such as a fall or direct blow



FIG 172 Rupture of biceps tendon. In this instance the rupture occurred at the supraglenoid tubercle. When the supinated forearm is flexed against resistance the biceps muscle forms a bulge in the lower part of the arm.



FIG 173 Rupture of biceps muscle at the lower musculotendinous junction. When the biceps muscle is contracted the muscle mass is displaced proximally to the upper part of the arm. Note the flattened area in the lower part of the arm below the bulge made by the contracted muscle.

to the arm or the shoulder may be the precipitating cause in some cases.

Site of Rupture. The site of rupture varies. In Gilcreest's series of 73 operative cases, 34 disclosed a rupture in the intracapsular portion of the tendon. The weakest portion of the tendon is the segment lying in the intertubercular sulcus just distal to its point of exit from the joint cavity. Less frequently the rupture may occur at the supraglenoid tuberosity at the musculotendinous junctions or within the muscle mass. In 9 of Gilcreest's 73 operated cases the tendon was avulsed from the supraglenoid tuberosity and in 22 the tear was located at the musculotendinous juncture, 20 being at the upper musculotendinous juncture.

In most cases there is total rupture of the tendon; occasionally there may be partial rupture. Meyer reported 2 partial ruptures of the tendon. With complete rupture the tendon may be found curled up at varying distances below the level of the intertubercular sulcus or it may bend upon itself like a 'jackknife'. In cases of partial rupture the torn fibers may reattach themselves to the bicipital groove (Fig 175).

Clinical Features. These vary and are

governed by such factors as the state of the tendon, whether it is normal or degenerated, the site of the rupture, whether the lesion is partial or complete and whether the tear is sudden or gradual. If the lesion occurs in a normal tendon or one exhibiting only minimal degenerative lesions there is, as a rule, a history of sudden contraction of the biceps muscle against some resistance followed by an audible snap and sharp lancinating pain in the shoulder which radiates to the anterior aspect of the upper arm. On the other hand, in a tendon already weakened by degenerative and attritional alterations, a rupture may occur without a

specific incidence of onset. Usually, these individuals give a history of vague distress, of weakness and of some impairment of function of the shoulder. These symptoms often parade under the erroneous diagnoses

size and the position of the swelling will depend upon the extent of the tear and the distance the muscle belly is permitted to be drawn away from the site of rupture. Rupture of the muscle belly allows the for

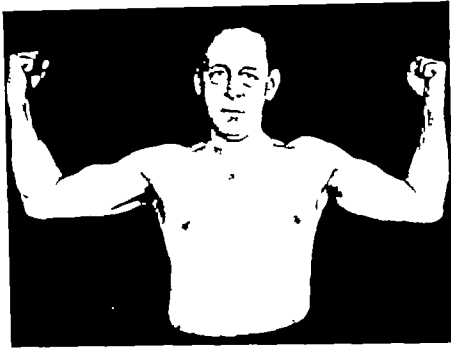


FIG 174 Rupture of the biceps causing no symptoms other than minimal weakness of the arm. In view of the absence of pain and little or no impairment of function, surgical intervention is not justifiable.

of arthritis, bursitis, tendinitis, sprains, etc.

The appearance of the upper arm varies according to whether the rupture of the tendon is complete or incomplete, and the site of the lesion. In complete lesions of the tendon the muscle mass will be drawn to a lower level, forming a soft swelling in the lower third of the upper arm when the forearm is extended; when the elbow is flexed the mass becomes firmer but does not reach the hardness of the biceps muscle on the normal side. The interval between the deltoid and the biceps muscle is increased (Fig 172). A complete rupture at the lower musculotendinous joint exhibits a muscle mass drawn upward in the upper third of the arm while the lower third appears flattened (Fig 173).

It is obvious that in partial ruptures the

formation of a hiatus between the two ends, its size being governed by the severity of the tear. In complete tears a wide gap is demonstrable when the forearm is flexed against resistance. In minor incomplete tears only a small sulcus is demonstrable.

No hematoma or ecchymosis results in high, acute ruptures if the lesion occurs through an avascular portion of the tendon; whereas in lesions through the belly of the muscle or at the musculotendinous junctions a large hematoma and discoloration of the anterior and the inner aspects of the upper arm may occur. Also in recent lesions pressure over the bicipital groove and over the muscle swelling elicits varying intensities of tenderness and pain which gradually subside until only some soreness remains. No significant tenderness is demonstrable in

old cases or in those resulting from degeneration of attritional changes

The tests of Vergason, of Ludington and of Hueter may be of some diagnostic value

VERGASON'S TEST When the elbow is flexed 90° and the forearm is pronated active supination of the forearm against resistance produces pain in the bicipital groove indicating lesions of the biceps tendon or its sheath or both. This is not a very dependable test

LUDINGTON'S TEST The patient places both hands with fingers interlocked and palms downward on the top of the head, with the weight of the extremities supported by the interlocked fingers. In this position the patient is made to contract and relax the biceps muscles. Active contraction can be palpated readily on the normal side but not on the affected side indicating a rupture or elongation of the tendon

HUETER'S TEST When the biceps muscle is relaxed flexion of the forearm is more forceful in pronation than in supination

Impairment of function depends in a large measure on the location and the extent of the lesion and the initiating causative agent. In acute traumatic ruptures of the tendon or the belly of the muscle severe impairment of function may result. pronounced weakness and pain may persist after the acute painful symptoms have subsided. On the other hand this lesion may cause insignificant disability in some individuals (Fig 174). Lesions with a gradual onset in elderly individuals and due to degenerative and attritional lesions seldom produce severe impairment of function. However some stiffness and soreness in the shoulder may be present

Treatment. Surgical repair is not always indicated in rupture of the biceps tendon. This is particularly true of latent lesions in elderly individuals whose impairment of function is not great and whose functional demands are diminishing. Conservative treatment of either partial or complete lesions is of doubtful value. If weakness and pronounced impairment of function exist

surgical intervention is indicated since it assures the patient of restoration of function in a relatively short period of time

The choice of procedure is governed by the type and the location of the lesion. If the rupture occurs in the intracapsular portion of the tendon, transplantation of the distal end of the tendon to the coracoid process gives excellent result, or, as Gilcreest recommended the tendon may be sutured to the short head of the biceps to the intertubercular groove to the pectoralis major or to the deltoid muscle

When the length of the tendon permits the author prefers to anchor it to the coracoid process as shown in Figure 170. If the tendon is short it is reattached to the intertubercular sulcus

Recent ruptures in the belly of the muscle or at the musculotendinous junctions are best repaired by deep interrupted mattress sutures of cotton or silk. If the suture line appears to be weak it may be reinforced by a fascial transplant. Old ruptures sometimes require excision of considerable scar tissue. The suture lines in these cases should be routinely reinforced by a fascial transplant. End to-end suture of the biceps tendon is done by the author only if the ends are healthy and readily approximated with minimum tension

Postoperative Management. The arm is immobilized in a plaster swathe for 3 weeks (Fig 198). Physical therapy is then begun to improve shoulder and elbow motion. Active flexion of the elbow is not permitted for 6 weeks. no forceful contractions are permitted before 10 weeks

TRAUMATIC DISLOCATION OF THE TENDON OF THE LONG HEAD OF THE BICEPS BRACHII MUSCLE

General Considerations. Varying degrees of displacement of the biceps tendon from the intertubercular groove was demonstrated in the specimens studied in the investigation on degenerative lesions of the shoulder joint. As first pointed out by Meyer the subluxation or dislocation of the

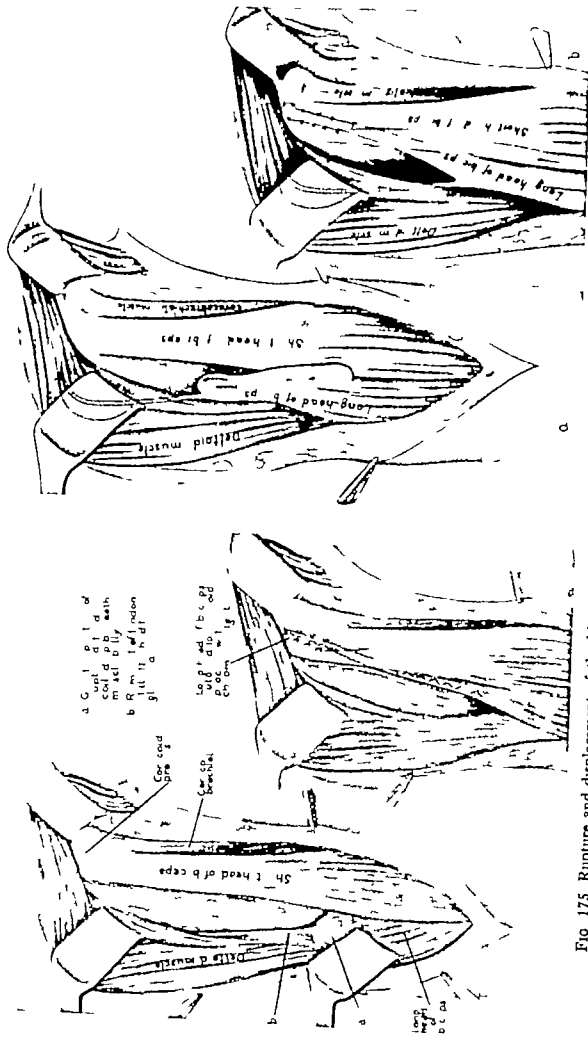


FIG 175 Rupture and displacement of the biceps tendon Rupture has occurred at the lip of the glenoid. (Gilcreest, Surg, Gynec & Obst 58 326)

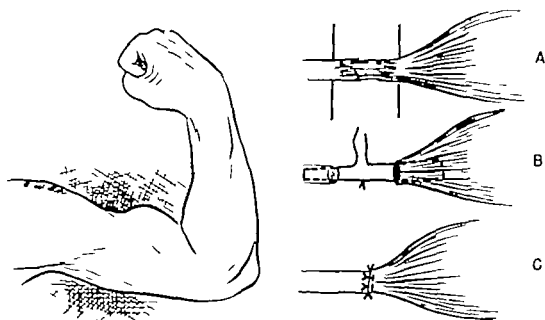


FIG 176 Technic of repair of rupture of the biceps tendon at the level of the upper musculotendinous juncture.

tendon was produced by gradual tearing away and elongation of the fibrous expansion of the insertion of the pectoralis major muscle and of the upper border of the subscapularis muscle from the anterior lip of the intertubercular sulcus. Hitchcock and Bechtol further pointed out that displacement of the tendon may be enhanced by the presence of a supratubercular ridge in the intertubercular sulcus.

All these observations have been confirmed in the study on degenerative lesions of the shoulder in Chapter 3. However, one is forced to agree with Abbott and Saunders that acute traumatic dislocations of the biceps tendon differ from those observed in shoulder joints of elderly individuals exhibiting advanced degenerative abnormalities. In traumatic lesions the presence of a supratubercular ridge facilitates the displacement of the tendon. Strenuous efforts with the arm in abduction and external rotation are responsible for the displacement, as a rule. In this position the tendon is forced against the lesser tuberosity. In the study on bicipital tenosynovitis 3 patients were encountered who could subluxate voluntarily the biceps tendon by ab-

ducting, and rotating the arm externally. In each instance a snap was heard and felt when the maneuver was executed. The resulting tenosynovitis associated with acute and chronic dislocation of the tendon plays a major role in the symptomatology present.

Clinical Features. Generally the syndrome is initiated by forceful abduction and external rotation movements of the arm. Occasionally displacement occurs repeatedly without a traumatic incident. In the acute traumatic cases the onset is followed by severe pain in the anterolateral aspect of the shoulder which is often projected to the anterior aspect of the upper arm. There is loss of power. Motion is restricted and painful, particularly in movements of abduction and external rotation and of forward flexion and abduction. A snap which is felt and often heard can be produced by having the patient bring the arm down from an elevated position in the coronal plane with the arm rotated externally. The snap is heard as the arm passes below the horizontal at which time the biceps tendon slips mesially over the lesser tuberosity. Once displacement has occurred recurrent dislocation of the tendon is the rule. In the

chronic forms, repeated displacement produces inflammatory changes in the tendon sheath mechanism, consistent with the tenosynovitis, causing pain and stiff shoulders

Treatment Surgical intervention is the proper treatment. The region is exposed adequately by a deltopectoral incision. Reconstruction of the fibrous covering of the

intertubercular sulcus is not practicable. Such a procedure, if feasible, predisposes to formation of adhesions between the tendon sheath and the bicipital groove. The method of choice is severing the tendon at the superior glenoid rim and suturing it to the coracoid process (Fig 170). As an alternative, the end of the tendon can be sutured to the bicipital groove.

BIBLIOGRAPHY

- Abbott L. C. and Saunders L. B. de C. M. Acute traumatic dislocations of the tendon of the long head of the biceps brachii: a report of six cases with operative findings. *Surgery* 6 817 840 1939
- Baer W. S. Operative treatment of subdeltoid bursitis. *Bull. Johns Hopkins Hosp* 18 282 284 1907
- Bruns P. V. A System of Practical Surgery. Vol. 1 p 119 Philadelphia, Lea 1904
- Codman, E. A. The Shoulder Boston, Thomas Todd, 1934
- Cotton, F. Subluxation of shoulder downwards. *Boston M & Surg J* 185 405 1921
- DeQuervain. Über eine Form von chronischer Tendovaginitis. *Corr Blatt f Schweiz Aerzte* 25 339 1895
- Dickson J. A. and Crosby E. H. Periarthritis of the shoulder: analysis of two hundred cases. *J.A.M.A.* 99 2252 225, 1932
- Duplay S. De la périarthrite scapulo-humérale. *Rev. frat. d. trav. de méd.* 53 226 1896 (Tr. M. Week 4 253 1896. *M. Press* 59 571 573 1900)
- Fairbank, T. S. Fracture subluxations of the shoulder. *J. Bone & Joint Surg* 30 B 454-460 1948
- Gilcreest E. L. Rupture of muscles and tendons. *J.A.M.A.* 84 1819-1822 1925
- Gilcreest E. L. Common syndrome of rupture, dislocation and elongation of long head of biceps brachii: analysis of one hundred cases. *Surg. Gynec. & Obst.* 58 322 1934
- Gilcreest, E. L. Dislocations and elongation of long head of biceps brachii. *Ann. Surg.* 104 118-138 1936
- Gilcreest E. L., and Albi P. Unusual lesions of muscles and tendons of shoulder girdle and upper arm. *Surg. Gynec. & Obst.* 68 903 1939
- Hitchcock, H. H., and Bechtol, C. O. Painful shoulder: observations of the role of the tendon of the long head of the biceps brachii in its causation. *J. Bone & Joint Surg* 30-A 263 1948
- Hueter C. *Arch. f. klin. chir* 5 321-323 1864
- King J. M. Jr and Holmes G. W. Diagnosis and treatment of four hundred and fifty painful shoulders. *J.A.M.A.* 89 1956-1961 1927
- Lans. *Beitr. z. klin. chir* 29 410 1901
- Leddenhose. Zur frage der rupture des biceps brachii, *Deutsche Ztschr. f. chir* 101 126 1909
- Lippmann, R. K. Frozen shoulder: periarthritis and bicipital tenosynovitis. *Arch. Surg.* 47 283 296 1943
- Ludington N. A. Rupture of long head of biceps flexor cubiti muscle, *Am. J. Surg.* 77 358 1923
- MacLaughlin H. L. Muscular and tendinous defects at the shoulder and their repair. *Instructional Courses* 1944. Ann Arbor Mich. Edwards 1945
- Meyer A. W. Unrecognized occupational destruction of the tendon of the long head of the biceps brachii. *Arch. Surg.* 2 130-144 1921
- Meyer A. W. Spontaneous dislocation of the long head of biceps brachii. *Arch. Surg.* 13 109 119 1926
- Meyer A. W. Chronic functional lesions of the shoulder. *Arch. Surg.* 35 646-674 1937
- Meyer A. W. Spontaneous dislocation and destruction of the tendon of long head of biceps brachii, *Arch. Surg.* 17 493-506 1928
- Meyer A. W. Unrecognized occupational destruction of tendon of long head of biceps brachii. *Arch. Surg.* 2 130 1939
- Meyer A. W. Quoted by Abbott & Saunders, *Surgery* 6 6 199 812 1939
- Mosely H. F. Rupture of supraspinatus tendon, *Canad. M.A.J.* 41 280 1939
- Mosely H. F. *Shoulder Lesions*, Springfield Ill. Thomas 1945 pp 58-65
- Mumford, E. B. and Martin, F. S. Calcified deposits in the subdeltoid bursitis. *J.A.M.A.* 97 690-694 1931
- Nevlaser J. S. Adhesive capsulitis of the shoulder. *J. Bone & Joint Surg* 27 211 222 1946

- Nichols E. H., and Richardson F. L. Arthritis Deformans J M Research 21 149-221 1909
- Painter C. F. Subdeltoid bursitis Boston M J 156 345 349 1907
- Parker F. Keefer C. S., Myer W. F., and Irwin R. L. Histologic changes in knee joint with advancing age Arch Path. 17 516-532 1934
- Pasteur F. La teno-bursite bicipitale J de radiol et d électrol. 16 419-426 1932
- Pasteur F. Sur une forme nouvelle de périarthralgie et d ankylose de l'épaule J de radiol et d électrol 18 327 328 1934
- Pasteur F. Ténobursite bicipitale Presse méd. 41 142 143 1933
- Schrager V. L. Tenosynovitis of the long head of the biceps humeri Surg., Gynec. & Obst 66 785 790 1938
- Stevens H. H. The action of the short rotators on the normal abduction of the arm with a consideration of their action on some cases of subacromial bursitis and allied conditions Am. J Med. Sc 138 870 1909
- Tarry J. M. Bicipital syndromes and their treatment New York State J Med. 46 996-1001 1946
- Wilson C. L. Lesions of supraspinatus tendon degeneration rupture and calcification Arch. Surg. 46 307 1943
- Yergason, R. M. Supination Sign J Bone & Joint Surg 13 160 1931

6

Calcareous Tendinitis of the Musculotendinous Cuff

SITE AND SEX

AGE

OCCUPATION

TRAUMA

INFECTION AND CONSTITUTIONAL DISEASES

PATHOGENESIS

PATHOLOGIC ALTERATIONS IN THE SUBACROMIAL BURSA AND MUSCULOTENDINOUS CUFF

RADIOGRAPHIC CHARACTERISTICS

TREATMENT

SUBACUTE AND CHRONIC SYNDROMES

OTHER THERAPEUTIC MEASURES

It is common knowledge that calcareous tendinitis is the most frequent cause of painful shoulders. Codman gave no actual statistics on the frequency of this lesion but was of the opinion that it was more common than any other affection involving the subacromial region. The author's statistics on 300 consecutive shoulder lesions disclosed calcareous tendinitis to be the etiologic factor in 43 per cent of the cases.

The term calcareous tendinitis is chosen because it more adequately conveys the basic pathologic process responsible for this syndrome. "Subdeltoid," or "subacromial bursitis with calcification," "bursitis calcuosa" and calcified deposits in the supraspinatus tendon portray a false pathologic concept of the entity. As will be shown subsequently the subacromial bursa is rarely if ever primarily involved. Reactionary changes in this structure always are secondary to an irritating focus in the adjacent tissues. Calcareous material may be observed in any portion of the musculotendinous cuff. Thus the designation "calcification in the supraspinatus tendon" may be a misnomer.

Calcareous tendinitis has been described

in relation to practically all tendons of the body. Next in frequency to the fibrotendinous cuff, it has been observed in the region of the greater trochanter and radiohumeral joint. The author also has encountered it in the tendo achillis and in the flexor tendon of the fingers. Cooper recorded the lesion in the metacarpophalangeal region.

SITE AND SEX

The lesion may occur in one or multiple areas of the cuff. In over 50 per cent of the affected shoulders it is observed as a single lesion involving the supraspinatus region of the cuff and encountered only rarely in the subscapularis tendon. Males are affected more frequently than females. The right shoulder is involved two times as often as the left. One half of the affected individuals reveal calcareous deposits in both shoulders.

AGE

Generally, calcareous tendinitis occurs in the fourth and the fifth decades but is observed occasionally in the latter part of the third decade. Rarely is it encountered after the sixth. The average age is 45 years.

OCCUPATION

Whereas ruptures of the cuff do occur in individuals doing laborious work, calcareous tendinitis is primarily a disease of persons employed in occupations, often sedentary, involving the use of the arms in slight abduction for hours at a time. Clerks, typists, stenographers and surgeons may be victims. As previously noted, the musculotendinous cuff is not mechanically suited to meet such abnormal stresses and strains, and if exposed to forces of this nature, attrition alterations ensue which accentuate the normal early degenerative changes that take place in this region during normal joint function.

TRAUMA

Excessive use of the arm which inflicts undue strain on the rotator cuff may be considered a form of trauma, and the accumulative effect may be responsible for advanced degenerative changes in the involved tissue. Calcification in the tendon fibers is evidence of nature's frustrated attempt to repair. It is reasonable to assume, however, that sudden trauma in the early and middle

age groups may be responsible for the tearing and the shredding of tendon fibers, followed by hemorrhage into the substance of the tendon, faulty repair and calcification.

A considerable interval must elapse between the initial trauma and radiographic evidence of calcification in the traumatized tendon. It is never demonstrable by radiographic examination made immediately after the injury. One case aged 32 was observed for 9 months before calcareous material was seen by radiographic examination. Excessive use in general and particularly occupations demanding overuse of the arm in abducted positions are predisposing. If not etiologic factors, sudden trauma may accentuate a subacute clinical stage of the syndrome or precipitate an acute syndrome, but it is not responsible for the calcareous tendinitis. It is generally known that calcified deposits in a tendon do not always produce symptoms. Many are quiescent and only accidentally discovered by the radiologist. Symptoms arise only when the sensitive peritendinous tissues are involved secondarily. One is led to conclude that the

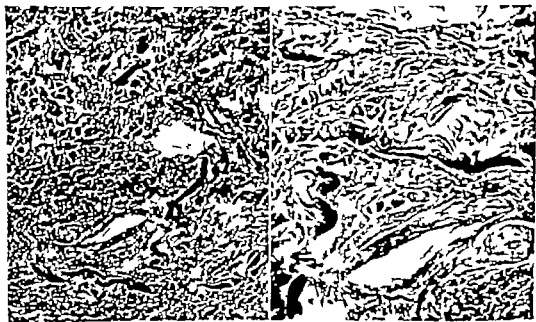


FIG. 177 (*Left*) Calcareous tendinitis in supraspinatus tendon responsible for a subacute syndrome. Advanced degeneration of tendon fibers is manifested by fragmentation of fibers and the presence of debris. Observe the presence of calcium deposits and round-cell infiltration ($\times 75$) (*Right*) The same section shown at left. ($\times 150$)

lesion precedes any sudden trauma which is immediately followed by the clinical features characteristic of this entity

INFECTION AND CONSTITUTIONAL DISEASES

Infection as a local or distant focus never has been proved to be an etiologic factor. Cultures and smears of the calcareous material never have disclosed a responsible organism. Associated distant foci of infection apparently play no part in the course of the disease. Patients with proved foci of infection (poor teeth infected sinuses, etc.) respond to therapy regardless of whether the focus was treated or not.

No relationship appears, therefore, between general disease and calcareous tendinitis. The only common denominator in persons affected with the latter is the factor of overuse of the arm in slightly abducted positions.

PATHOGENESIS

Many concepts have been formulated relative to the etiology and the mechanism of developing amorphous calcium deposits in

tendons. As yet the problem remains obscure. However, there are certain features on which there is more or less general agreement. Calcareous deposits are common findings in areas of necrosis of degenerated tissues. It has been proved conclusively that degenerative changes occur early in the musculotendinous cuff because this structure is not mechanically and anatomically adequate to meet the demands of a prehensile extremity. The point of greatest strain is in the region of the supraspinatus tendon, hence, degenerative lesions are encountered more frequently in this area than elsewhere in the cuff. Calcareous deposits in this region are found just proximal to the insertion of the conjoined tendon. Codman designated this area as the 'critical zone'—an area which sustains the maximum degree of strains and discloses the most severe degenerative changes. It is also the area through which primary transverse ruptures of the cuff occur most often.

Generally it is believed that the process begins with degeneration of the central tendon fibers in the "critical zone." This observation is substantiated by the findings in

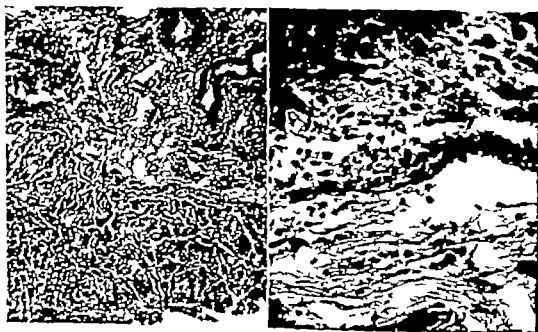


FIG 178. (Left) Calcareous tendinitis in supraspinatus tendon causing an acute syndrome. Observe degeneration of tendon fibers, calcium deposits, large blood vessels and infiltration of the tissue by leukocytes and round cells. ($\times 67$) (Right) A section of the area depicted at left. ($\times 267$)

autopsy specimens of small degenerated areas within the substance of the tendon having no communication with the joint cavity or the subacromial bursa. Small degenerated necrosed bits of tendon fibers were often discernible in these areas. Codman believed that the calcareous deposits found in the living are nature's attempt to heal these lesions.

Considerable controversy still exists relative to the actual mechanism of calcium deposition in degenerated tissue. Howland and Wells have offered a plausible theory which is applicable to lesions observed in the musculotendinous cuff. It may be summarized as follows: Calcium and inorganic phosphorus are held in solution in the blood serum in much higher concentration than would be possible in water because of high carbon dioxide tension and colloids in blood serum. Furthermore a high carbon dioxide tension is maintained by cellular activity; hence no precipitation of salts occurs from intercellular fluids. However, degenerated or dead tissue lowers the carbon dioxide

tension (or produces a relative state of alkalinity), thus favoring precipitation of calcium carbonate or calcium phosphate, or both, in a molecular combination. This is a local physiochemical process. Patients having calcareous tendinitis but no other disease have normal blood calcium levels.

PATHOLOGIC ALTERATIONS IN THE SUBACROMIAL BURSA AND MUSCULOTENDINOUS CUFF

The character of the pathologic changes in the subacromial bursa and affected region of the cuff depends upon the size, the location and the consistency of the calcareous material and the severity of the inflammatory reaction around it. As previously stated calcareous deposits form within the substance of the tendon. When they are discrete localized lesions, not irritating the floor of the bursa, there may be no symptoms present. They are more or less quiescent and may remain so for many years. Frequently such lesions are discovered accidentally by the radiologist.



FIG 179 (Left) Section of calcareous tendinitis in subscapularis tendon causing an acute syndrome. Note numerous areas of calcium deposits, pronounced infiltration of round cells and leukocytes, along with advanced degeneration of tendon fibers. ($\times 67$) (Right) Subacromial bursa in case depicted at left. Observe increased vascularity, pronounced infiltration of leukocytes and round cells, and edema and thickening of the bursal wall ($\times 134$)

At this stage, no changes are discernible in the bursa. Usually the deposits enlarge gradually until they reach the floor of the bursa. In this position they may cause pain on abduction and elevation or rotation of the arm. Symptoms are the result of mechanical impingement of the lesion against the edge of the coraco-acromial arch during the above arcs of motion. Frictional trauma of this nature is responsible for secondary changes in the floor of the bursa. Now the deposit appears as a well-defined mild swelling with a white center, not under tension. The floor of the bursa may be thickened and covered with numerous villi of varying sizes.

Incision of the tumefaction discloses a whitish, gritty material incorporated within the tendon fibers. Removal can be achieved only by curettage or excision of the entire area. If curetted degenerated tendon fibers are noted in the gritty material. Often a fair sized cavity remains, the borders of which are comprised of shredded, degenerated stubs of tendon fibers. Lesions just described do not ordinarily give rise to acute symptoms. Clinically, they are considered responsible for mild symptoms only. Continued frictional trauma or sudden strains may accentuate the intensity of the symptoms giving rise to a subacute or an acute syndrome, depending upon the intensity of the clinical features.

Lesions responsible for the subacute phase lie directly under the bursal floor, which is stretched tightly over the calcareous mound. The swelling has a whitish or yellow center surrounded by a circular bluish zone of congested synovial membrane. In cases of long standing or repeated aggravation, the floor of the bursa may be thickened and studded with fine synovial villi. Adhesions within the bursa are not infrequent findings in this stage.

The size of the calcareous mass varies from a few millimeters to 5 or 6 cm. In length the average size encountered is about 1½ to 2 cm. Upon incision of the top of the mound there escapes a whitish or

yellow material which has the consistency of tooth paste. Occasionally, gritty material is found in the contents. In long standing cases the deposit is incorporated in the tendon fibers and does not flow out of a distinct cavity in the substance of the tendon but must be evacuated with a curette. Strands of degenerated tendon fibers are discernible in the calcareous material.

During the acute stage the pathologic changes are similar to but of greater intensity than those in the subacute stage. If the floor of the bursa has ruptured, varying amounts of a milky fluid, composed of calcareous material and serum, are encountered upon opening the bursa. If the floor is intact, serous fluid escapes from the bursa, and the bursal floor may be covered with a film of fibrin.

The deposit has a whitish center and is surrounded by a deep-red or violet congested circular zone, the synovial floor of the bursa is stretched tightly over the swelling, which appears to be under great tension. If the top of the tumefaction is nicked with a scalpel a milky fluid escapes. There appears to be a parallelism between the acuteness of the symptoms and the consistency of the calcareous material.

Symptoms in the acute stage are the result of irritation and stretching of the sensitive floor of the bursa. When tension is released the symptoms subside.

CLINICAL FEATURES

Pain, muscle spasm, restriction of motion, and muscle atrophy are the cardinal features of calcareous tendinitis. These symptoms differ greatly in intensity and severity, permitting one to recognize, clinically three distinct variations of the same syndrome: the chronic, the subacute and the acute syndrome. But such a clinical classification must not be adhered to rigidly, since many cases are borderline in character. Others present all three stages at different times.

SUBACUTE SYNDROME

Most patients with calcareous tendinitis

disclose more or less subacute clinical manifestations. As a rule, the onset is insidious, the patient first feeling a sudden twinge of pain or a catch in the shoulder during certain motions particularly on abduction and internal rotation.

Pain results from impingement of the sensitive irritated area in the musculotendinous cuff against the acromion or coraco-acromial ligament. After it has passed under these structures, the pain disappears. This explains why many patients find relief in the early stages when they sleep with the hand behind the head. As the pain becomes more severe the patient instinctively avoids motions which produce pain and uses the arm only within the painless arcs. In mild cases the syndrome may remain static for many months and then it may disappear. On the other hand the pain may increase in intensity and even become excruciating at the level of impingement. Pain projected to the insertion of the deltoid is a constant observation. In severe cases there may be pain referred posteriorly to the inferior angle of the scapula and up the neck to the suboccipital region.

Occasionally in the severer forms it radiates to the back of the forearm and to the fingers particularly to the thumb and the index finger. The hand may be puffy and the fingers swollen and painful. Generally it is more severe at night the patient has great difficulty in finding a comfortable position for the arm. He is unable to lie on the affected side. Excruciating tenderness usually can be elicited by pressure over the inflamed bursa also pressure over the insertion of the deltoid produces pain in most instances. At times a small tender mass which rotates with the humerus can be palpated over the greater tuberosity.

In the early stage spasm of the rotator muscles is not a prominent feature being intermittent or absent however with progression in severity of the lesion muscle spasm becomes pronounced and constant. The glenohumeral motion is at first markedly restricted and finally obliterated com-

pletely. Abduction and elevation are now barely possible to the horizontal plane and take place entirely in the scapulothoracic joint, normal scapulohumeral rhythm is lost. Muscle atrophy of the spinatus and the deltoid muscles is also an outstanding feature. The patient now presents a typical clinical picture of frozen shoulder.

It is interesting to note that as the amount of restriction of motion increases, the pain decreases. Many cases with little or no glenohumeral motion are practically free of pain.

It is apparent that calcareous deposits like bicipital tenosynovitis, may initiate in inflammatory changes in the rotator cuff which terminate in frozen shoulder. As a rule this process never is permanent. With absorption of the calcareous deposits and healing of the defect in the floor of the subacromial bursa the entire process is reversed adhesions are stretched and absorbed and motion returns.

Fortunately most subacute cases do not run such a violent course. As a rule the course is more clement and complete recovery can be anticipated in a relatively short period of time, if the case is treated adequately. Some patients have comparatively little pain spasm and restriction of motion are the predominant features from the very onset of the syndrome. On the other hand while other patients disclose very little spasm and limitation of motion, the pain may be intense and constant.

ACUTE SYNDROME

Acute syndromes run a relatively short fulminating course. Generally there is a sudden onset with no previous clinical evidence of any pathologic disorder in the cuff. At times the attack is precipitated by overuse of the arm or by trauma or it appears during the course of a subacute or chronic phase. The characteristic features are sudden intense pain on any attempts at motion, with localized tenderness and severe spasm of the rotator muscles.

Agonizing pain may appear suddenly dur-

ing the night. The slightest motion accentuates the pain, which is projected to the deltoid insertion and even to the fingers. The usual history is one of sleepless nights, agonizing pain and failure to obtain relief from the usual sedatives. In untreated cases, such symptoms may persist for a week or two and then they subside. Spasm becomes less severe, and restoration of motion is achieved gradually. In some cases, varying degrees of spasm and restriction of motion in the glenohumeral joint may persist for many months following the acute episode. Although the interval of time before complete restoration of motion is unpredictable, full recovery is a certainty.

The sudden onset is the result of an acute inflammatory process involving the floor of the bursa. At this stage the calcareous mass is under great tension; it is surrounded by a red, congested zone of synovial tissue. Frictional trauma accentuates the pain and produces spasm of the rotators and fixation of the glenohumeral joint. Fibrin and a serous exudate pour into the bursa. Should the floor rupture, a milky fluid escapes into the bursa. Thereupon pain subsides, as all tension is relieved. Nature readily absorbs the calcified material evacuated into the bursa. If there is complete absorption of the calcareous material, healing ensues and no recurrence is to be anticipated. However, if some calcified material remains, there is always the possibility of recurrent attacks of an acute syndrome or the lesion may pursue a subacute or chronic course.

CHRONIC SYNDROME

Symptoms are very mild in these cases. The only complaint may be a sudden catch or slight twinge of pain on internal rotation or elevation of the arm. There is no muscle spasm or restriction of motion. Pain is the result of mechanical impingement of the calcareous mass against the acromion or the falciform edge of the coraco-acromial ligament during the above-mentioned motions. Such a syndrome may persist for years without much apparent disability.



FIG. 180 Dense, discrete, calcareous deposit, giving rise to no symptoms except an occasional twinge of pain when the arm is abducted.

However, at any time (especially if the factor of trauma or excessive use is added) the lesion may pursue a subacute or acute course.

RADIOGRAPHIC CHARACTERISTICS

Calcareous deposits range in length from a few millimeters to 5 or 6 centimeters. Although their configuration is considerably variable, at first the mass is usually in the long axis of the cuff fibers. Later, with rupture of the floor of the subacromial bursa and dispersal of the calcium particles, an irregular crescent or globular mass may be discernible. Generally, in long-standing cases, in either the chronic or the subacute stage, the deposits are more or less discrete and homogeneous. The density may be uniform throughout the entire mass or it may be spotty because of small areas of varying

density scattered throughout the calcareous deposit (Fig 180) In the acute stage the mass is less dense and more spotty It may be amorphous and fluffy in character (Fig 181)

Roentgenographic visualization and precise localization of the deposits may at times prove to be rather difficult Routinely, these films should be taken in different positions—in external rotation in internal rotation and at the midposition (Fig 182) In addition Bosworth advocates routine fluoroscopic examination and the taking of spot films The value of such a procedure in some areas is obvious but as a routine measure it is superfluous

TREATMENT

Treatment in all phases of calcareous tendinitis should be tempered by the knowledge of the following clinical observations

- 1 The disease is self limiting

- 2 All patients, sooner or later, make a complete recovery, with no residual complications

- 3 Nature produces relief of symptoms by rupturing the calcareous deposit and allowing it to escape into the bursa where it is absorbed rapidly, or by gradual absorption of the deposit in situ

- 4 Complete removal or absorption of the calcareous material results in a permanent cure Incomplete removal may predispose the patient to recurrences.

- 5 Recovery is the rule—regardless of the method of treatment, in spite of some treatment and without treatment

ACUTE SYNDROME

During the acute phase pain can be relieved either by aiding nature in evacuating the deposit into the bursa where it will become absorbed or by reducing the inflammatory process in the floor of the bursa



FIG 181 (Left) Calcareous deposit in the supraspinatus tendon producing a subacute syndrome Note the irregular small areas of varying density scattered throughout the mass. (Right) Small calcareous deposit in supraspinatus tendon producing an acute syndrome Observe the amorphous nature of the mass also note that it has ruptured into the bursa. At operation a white milky serous fluid was found in the subacromial bursa.

Rupture of the deposit can be achieved by one of several methods, such as (1) surgical removal, (2) aspiration and needling, and (3) irrigation of the bursa and manipulation. The intensity of the inflammatory process is best reduced by radiotherapy.

SURGICAL REMOVAL OF THE CALCAREOUS DEPOSIT

Although operative intervention may appear to be radical, relief from pain is prompt and certain. The restoration to normalcy is more rapid than in the other two methods indicated above, and nonrecurrence is assured. These advantageous features should be offered to the patient if counterindications to surgery do not exist.

An anterior deltoid splitting incision is simple and provides adequate exposure of the subacromial bursa. The patient is placed on the table in a sitting position with a sand bag under the affected shoulder and another under the corresponding hip. By drawing the patient close to the edge of the table the elbow may be dropped below table level,

thereby making the tuberosity more prominent. The hand and the forearm on the affected side are draped so that the arm may be maneuvered freely during the operation.

The skin incision begins immediately below the acromioclavicular joint and extends distally on the anterior aspect of the arm for from $2\frac{1}{2}$ to 3 inches. The incision is deepened through the full thickness of the anterior portion of the deltoid by blunt dissection, exposing the roof of the bursa. While the forearm is held vertical to the table, the roof of the bursa is incised, the opening being directly over the bicipital groove. All regions of the bursa can be visualized readily merely by rotating the arm. No difficulty is encountered in locating the calcareous deposit if it is beneath the floor of the bursa in the supraspinatus region of the cuff.

Visualization of the mass may be slightly more difficult if it lies in the subscapularis region or in the extreme end of the infraspinatus or in the teres minor regions. The



FIG 182 (Left) Calcareous deposit in the subscapularis tendon. Observe its relation to the glenoid brim with the arm in internal rotation. (Right) Same shoulder with the arm in external rotation. Observe how the position and configuration of the mass has altered as compared to that shown at left.

tumefaction is incised parallel with the cuff fibers and its contents are evacuated with a curette. Occasionally, the necrotic walls of the remaining cavity are excised by elliptical incisions parallel with the axis of the tendon fibers. The cavity is irrigated thor-

oughly with normal saline solution. If the defect is extensive, it is closed by side-to-side sutures.

A thorough search for the presence of any other deposits should be made and any suspicious areas should be nicked with the



FIG 183 (*Top left*) Calcareous deposit producing an acute fulminating syndrome treated by aspiration and needling (*Top right*) Roentgenogram taken the day after aspiration and needling of calcareous mass depicted at left. Observe that not all calcified material was removed (*Bottom*) Roentgenogram of the shoulder depicted above 4 weeks later. Note the complete absorption of calcareous material. The patient achieved complete restoration of function.

point of a scalpel to determine the existence of hidden calcareous masses. Multiple punctures of the cuff adjacent to the lesion ensure an active hyperemia which favors absorption of any remaining calcareous material. To facilitate free excursion of the humeral head under the coraco-acromial arch, the coraco-acromial ligament is divided routinely at its insertion into the medial edge of the acromion, and as much of the bursal sac as is accessible is removed.

For the first 24 hours following operation the arm is suspended to an overhead frame with lateral traction. On the second day gravity free pendulum exercises are started on a schedule of every three hours for from 3 to 5 minutes. The range of motion is maintained at all times within the patient's tolerance of pain. As the acute symptoms subside, exercises are done every hour, and wall-crawling and pulley exercises are added.

Physical therapy should supplement this regime. Radiant heat and gentle massage to the shoulder girdle and the upper arm add to the comfort of the patient. In most instances full restoration of painless motion is attained in 3 weeks although some few cases (complicated with pronounced fixation of the glenohumeral joint prior to operation) may require from 6 to 8 weeks. It is interesting to note that the more acute the syndrome prior to operation the sooner it will recover. This observation of course is true of all therapeutic measures employed.

ASPIRATION AND NEEDLING

If surgery is contraindicated or refused aspiration and needling are advocated. Although in many instances relief from pain is as dramatic as when surgical intervention is employed it is a more-or-less blind procedure. If more than one deposit is present one is certain to fail to rupture and evacuate all of them.

The patient assumes the same position on the table as that described for open operation. After the point of maximum tender-

ness is located and marked, the arm is prepared surgically and draped as meticulously as when an open procedure is to be done. With a hypodermic needle containing a 1 per cent solution of procaine a small wheal is raised in the skin over the designated point of maximum tenderness. A larger-gauge needle is used to infiltrate the subcutaneous tissue, the muscle and the roof of the bursa. Anesthesia of the area having been attained, an 18-gauge needle is then directed toward the calcareous mass, which in some instances is distinctly palpable. As the needle penetrates the calcified area a definite sensation of grating is felt. Upon withdrawal of the plunger, some fine flaky, calcareous material will be aspirated into the syringe.

Barbotage is continued at various levels in the calcareous mass until all accessible calcined particles are removed. This is indicated by the return of clear fluid into the syringe. A 1 per cent solution of procaine is used throughout the procedure (Fig 184).

To assure absorption of any remaining particles the entire area is multiple-punctured with a large straight skin needle. The adjacent areas, as well as the site of the calcareous mass, are punctured thoroughly. An active hyperemia is produced, which dissolves and absorbs the calcareous material left *in situ*. Puncturing also allows some of the particles to escape into the bursal sac where they are absorbed readily (Fig 183).

AFTER TREATMENT

For the first 24 or 48 hours following aspiration and needling the patient should be kept under sedation. Hot packs should be applied to the shoulder. If possible the arm should be suspended from an overhead frame and lateral traction should be applied. The patient must be encouraged to move the arm several times a day through a normal range of motion.

When the patient arises from bed gravity free pendulum exercises are begun with wall-crawling and pulley exercises added.

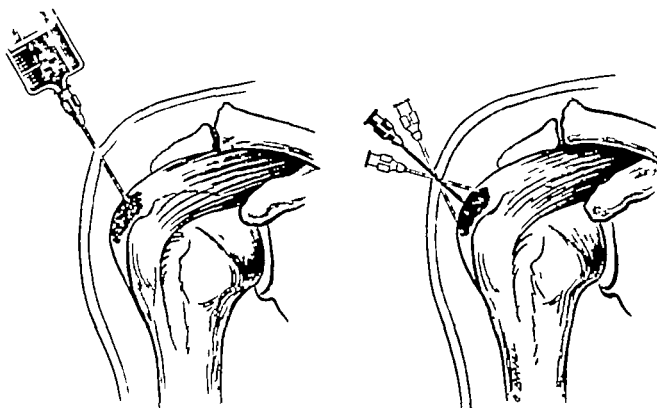


FIG 184 Aspiration and needling of the calcareous deposit in the supraspinatus tendon.

later Motion should be executed within a painless arc. As sensitivity diminishes, the arc of motion is increased. Radiant heat and gentle massage may be added to this regimen.

Occasionally one fails to aspirate calcareous material. In such instances, multiple-puncture of the area as described above is performed. Rupture of the floor of the bursa will relieve tension and allow calcified particles to disperse into the bursa where they will be absorbed. Absorption of the calcareous mass is still further enhanced by the hyperemia which follows the puncturing. Although this procedure is not as efficacious as aspiration and needling it will alleviate the acute symptoms in most instances.

IRRIGATION

Patterson and Darrach described a technic of irrigation of the subacromial bursa as a result of which they recorded complete relief of symptoms in 57 of 63 cases with an average economic disability of 4.8 days.

Essentially the method comprises continuous irrigation of the bursa with two 18-gauge needles. Normal saline is forced out of one needle and through the other. Washing of the bursa continues until the fluid flowing out of the second needle is clear (usually from 30 to 60 cc of saline solution is used). Following irrigation the patient is encouraged to move the arm in all directions. Occasionally gentle manipulation is employed to break up adhesions in long-standing cases. After treatment is essentially the same as that following aspiration and needling.

MANIPULATION

Some workers occasionally manipulate the shoulder during the acute stage. The author does not advocate this method except when the purpose of the manipulation is rupture of the deposit. Once the floor of the bursa overlying the calcareous mass is ruptured and the mass dispersed into the bursa relief of symptoms is assured and absorption of the calcium follows.

ROENTGEN RAY THERAPY

There is general agreement that roentgen radiation is a valuable method of treatment during the acute phase of some cases. Some workers claim unusual dramatic cures. Isidore Lattman recorded that patients were relieved of acute symptoms and restored to normalcy more rapidly by this method than by any other relief being obtained in 13 of 20 cases within from 24 to 48 hours. However Sandstrom reported that of 73 cases more than half were treated for from 2 to 5 weeks, a quarter for from 2 to 4 months and a few for over a year.

The author has seen some quick dramatic cures following irradiation therapy. In some instances however pain was greatly accentuated after treatment, while recovery was slow in others. Several patients had recurrences. Therefore roentgen ray therapy is advocated only when operative intervention and aspiration and needling are contraindicated or refused. It is believed that irradiation produces relief of symptoms by decreasing the intensity of the inflammatory process thereby reducing the tension beneath the floor of the bursa.

SUBACUTE AND CHRONIC SYNDROME

Calcareous deposits in the subacute and chronic phases vary greatly in texture ranging from a homogeneous tooth paste consistency to discrete circumscribed dense masses (Figs 180 and 181 *left*). In more acute cases the masses tend to assume an amorphous character. In the absence of pronounced inflammatory changes the symptoms are produced by mechanical impingement of the calcareous deposit against the coraco-acromial ligament or acromion. As previously noted both the subacute and the chronic phases may be transformed into an acute syndrome gradually or suddenly and with or without provocation.

Management is similar to that described for the acute stage. Surgical removal and needling assure the patient a more rapid and

more nearly complete recovery. Inasmuch as the lesions may be well circumscribed and the symptoms not too pronounced aspiration and needling or needling alone may produce the desired results. Whatever the choice of treatment it should be supplemented by such intensive physical therapy as gravity free pendulum exercises wall crawling and pulley exercises.

This is particularly true in long standing cases or in cases with repeated attacks of acute exacerbations of pain in which there may be varying degrees of restriction in the glenohumeral joint with contractures and shortening of the internal rotator and adductor muscles.

Roentgen-ray therapy is believed by some workers to give satisfactory results in subacute cases. However it is of doubtful value in the chronic cases. As in the acute form, pain may be intensified after the first or second dose making sedation and analgesic medication necessary. Unless the pain is too intense the patient is encouraged and urged to follow the prescribed regimen of exercises previously described to restore normal range of glenohumeral motion.

Following roentgen therapy a certain number of cases will reveal on roentgenograms a definite diminution of the calcareous deposit and occasionally total resorption of the mass. In most instances, however there will be little or no perceptible change. These same observations have been noted in untreated cases. Thus it is difficult to say whether or not roentgen ray therapy has any specific effect upon the calcareous mass. However it is a clinical fact that as long as roentgenographic evidence of calcareous tendinitis exists the patient is likely to develop symptoms of varying severity. It is obvious that the most desirable method of treatment is one which will result in complete dispersal and resorption of the calcareous material.

Manipulation in subacute and chronic forms should have only one objective rupture of the deposit. If this does not appear feasible nothing will be gained by this

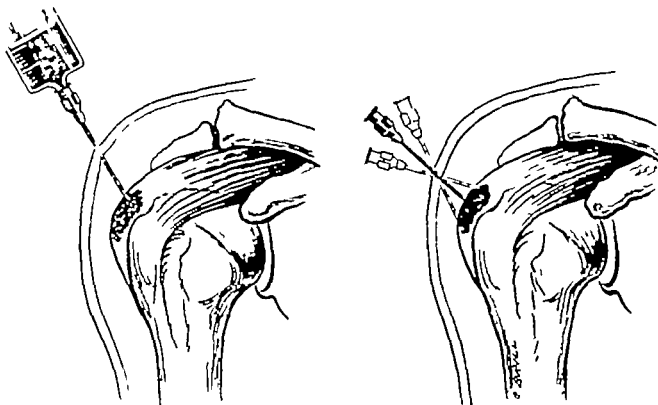


FIG 184 Aspiration and needling of the calcareous deposit in the supraspinatus tendon.

later Motion should be executed within a painless arc. As sensitivity diminishes the arc of motion is increased Radiant heat and gentle massage may be added to this regimen

Occasionally one fails to aspirate calcareous material In such instances multiple-puncture of the area as described above is performed Rupture of the floor of the bursa will relieve tension and allow calcified particles to disperse into the bursa where they will be absorbed Absorption of the calcareous mass is still further enhanced by the hyperemia which follows the puncturing Although this procedure is not as efficacious as aspiration and needling it will alleviate the acute symptoms in most instances

IRRIGATION

Patterson and Darrach described a technique of irrigation of the subacromial bursa as a result of which they recorded complete relief of symptoms in 57 of 63 cases with an average economic disability of 4.8 days

Essentially the method comprises continuous irrigation of the bursa with two 18-gauge needles Normal saline is forced out of one needle and through the other Washing of the bursa continues until the fluid flowing out of the second needle is clear (usually from 30 to 60 cc. of saline solution is used) Following irrigation the patient is encouraged to move the arm in all directions Occasionally gentle manipulation is employed to break up adhesions in long standing cases After treatment is essentially the same as that following aspiration and needling

MANIPULATION

Some workers occasionally manipulate the shoulder during the acute stage The author does not advocate this method except when the purpose of the manipulation is rupture of the deposit. Once the floor of the bursa overlying the calcareous mass is ruptured and the mass dispersed into the bursa relief of symptoms is assured and absorption of the calcium follows.

- irradiation in the treatment of inflammation
Am. J Roentgenol 45 74 106 1941
- Sandstrom, C. Peritendinitis calcarea a common disease of middle life its diagnosis pathology and treatment, Am. J Roentgenol 40 1 1938
- Wells H G Calcification and ossification Arch. Int. Med 7 721 1911
- Wells H G Calcification and Ossification Harvey Lectures New York 1910-11 p 102
- Wilson P D The painful shoulder Brit M J 21 1261 1939

method of treatment. In the presence of advanced periarthritic adhesions and contractions of the internal rotators and adductors of the shoulder, severe damage may follow manipulation. Tearing and violent stretching of adhesions result invariably in more adhesions also, in long standing cases which have pronounced demineralization of the humerus, fracture of the surgical neck is not an infrequent occurrence. The author has seen two such tragedies.

Frozen shoulders associated with calcareous tendinitis should be managed in the same manner as frozen shoulders initiated by bicipital tenosynovitis. Once the entity is established the bicipital tendon and sheath always are involved. After removal of the deposits recovery will be expedited if the biceps tendon is removed from the groove and reattached to the coracoid process. (See Treatment of Frozen Shoulder.)

OTHER THERAPEUTIC MEASURES

Diathermy is of value only in that it in-

duces hyperemia or increases an existing inflammatory process, thereby favoring rupture of the calcareous mass and the alleviation of pain. It has no specific therapeutic value and may increase the intensity of the symptoms until rupture of the bursa occurs. After rupture of the floor of the bursa it may accelerate absorption of the calcium particles. The same may be said of hot packs which are a valuable adjunct following aspiration and needling or needling alone.

Cold packs are definitely indicated during the acute or the subacute stage. They tend to mitigate the inflammatory process and, hence to reduce the tension beneath the sensitive synovial floor of the bursa.

Injection of procaine solution (5 cc.) into the point of maximum tenderness without aspiration or needling sometimes gives dramatic relief. In such instances rupture of the calcareous deposit into the bursa is undoubtedly induced as a result of this procedure.

BIBLIOGRAPHY

- Bosworth, B. M. Calcium deposits in the shoulder and subacromial bursitis: a survey of 1212 shoulders. *J. A. M. A.* 116:2477, 1941.
- Bosworth, B. M. Examination of shoulders for calcium deposits. *J. Bone & Joint Surg.* 23:567-577, 1941.
- Boyd, H. B. Affections of Muscles, Tendons and Tendon Sheaths. *Campbell's Operative Orthopedics*, pp. 1212-1262. St. Louis: Mosby, 1949.
- Breckner, W. M. Prevalent fallacies concerning subacromial bursitis: its pathogenesis and rational operative treatment. *Am. J. M. Sc.* 149:351, 1915.
- Codman, E. A. *The Shoulder*. Boston: Thomas Todd Co., 1934.
- Cooper, W. Calcareous tendinitis in metacarpophalangeal region. *J. Bone & Joint Surg.* 24:114, 1942.
- DePalma, A. F. Calcareous deposits in soft tissues about the proximal interphalangeal joint of the index finger. *J. Bone & Joint Surg.* 29:808, 1947.
- Elmorille, R. C. Calcareous deposits in supraspinatus tendon. *Brit. J. Surg.* 20:190, 1932.
- Goldenberg, R. R. Leventhal, G. C. Supra-trochanteric calcification. *J. Bone & Joint Surg.* 18:205, 1936.
- Howland, V. Etiology and pathogenesis of rickets. *Medicine* 2:349, 1923.
- Howorth, M. B. Calcification of tendon cuff of shoulder. *Surg. Gynec. & Obst.* 80:337, 1945.
- Lattmann, I. Treatment of subacromial bursitis by roentgen irradiation. *Am. J. Roentgenol.* 36:55, 1936.
- McLaughlin, H. L. Lesions of the musculotendinous cuff of the shoulder: observations on the pathology, course and treatment of calcific deposits. *Ann. Surg.* 124:354, 1946.
- McLaughlin, H. L. Muscular and Tendinous Defects at Shoulder and Their Repair. *Am. Acad. Orth. Surgeons: Reconstruction Surgery of the Extremities*. Ann Arbor: Edwards, 1944.
- Painter, C. F. Subdeltoid bursitis. *M. & S. J.* 156:345, 1907.
- Patterson, R. L. Jr. and Darrach, W. Treatment of acute bursitis by needle irrigation. *J. Bone & Joint Surg.* 19:993, 1937.
- Pendergrass, E. P. and Hodas, E. J. Roentgen

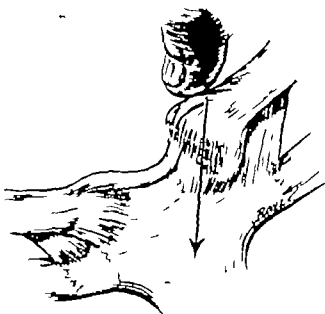


FIG 185 Dislocation of sternoclavicular joint. Reduction is readily achieved by pulling shoulders upward and backward or by making direct pressure over the sternal end of the clavicle. Maintenance of the reduction is difficult.

The most popular method is the posterior figure-of-eight bandage. Plaster-of-Paris bandage is more effective than linen or muslin bandage. A large wad of cotton is placed in each axilla, and a small piece of felt in front of each shoulder. Both shoulders are then pulled upward, outward and backward. While the position is being maintained, a posterior figure-of-eight plaster-of-Paris bandage four inches wide is applied tightly around both shoulders. The bandaging should be snug enough to maintain the shoulders upward and backward but should not embarrass the circulation of the arms.

Before the plaster-of-Paris bandage becomes hardened, the patient is placed in a recumbent position with a folded sheet between both shoulders. The operator then makes downward pressure on the front of both shoulders and holds the position until the bandage is sufficiently dry and hardened to maintain the shoulders in the described position. The dressing is worn for six weeks. Should the arms become cyanotic, the patient is instructed to pull back voluntarily on the shoulders and abduct the arms from the side of the body (Fig 186).

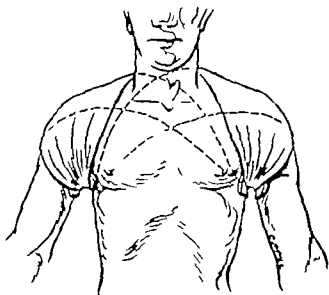


FIG 186 Posterior figure-of-eight plaster-of-Paris bandage, 4 inches wide, holding shoulders upward and backward. Note cotton wadding in each axilla.

Some operators prefer to use the clavicular cross such as is used for fractures of the clavicle. This, too, adequately maintains the shoulders in the desired position while healing occurs.

Should the above methods fail, the patient may be treated in bed with the arm in lateral traction (90° to 120° abduction) and a sand bag over the sternoclavicular joint (Fig 187). This position reduces the dislocation and maintains reduction. It is retained for three weeks, which period permits sufficient healing of soft tissue to stabilize and prevent redisplacement of the proximal end of the clavicle when the traction apparatus is removed. A posterior figure-of-eight plaster dressing is then applied, and the patient is made ambulatory. The dressing is worn for from three to five additional weeks. Surgical intervention is rarely indicated in fresh traumatic dislocations. Adhesive dressings making pressure over a piece of felt placed over the sternal end of the clavicle are not satisfactory. The adhesive strips tend to stretch and become detached thereby allowing redisplacement to occur.

Chronic (Old) Dislocations. Not all old dislocations demand treatment. In many

Dislocations of the Shoulder Joint

DISLOCATIONS OF THE STERNOCLAVICULAR JOINT

DISLOCATIONS OF THE ACROMIOCLAVICULAR JOINT

DISLOCATIONS OF THE SCAPULOHUMERAL JOINT

COMPLICATIONS OF DISLOCATIONS OF THE SHOULDER

OPEN REDUCTION FOR INITIAL DISLOCATION
LUXATIO ERECTA

ACUTE TRAUMATIC POSTERIOR DISLOCATION
OLD DISLOCATIONS

RECURRENT DISLOCATION OF THE GLENO-
HUMERAL JOINT

POSTERIOR RECURRENT DISLOCATION

DISLOCATIONS OF THE STERNOCLAVICULAR JOINT

Lesions of the sternoclavicular joint are not encountered frequently. They may result from falls on the point of the shoulder or in accidents in which great force is applied to the tip of the shoulder from above for example being struck by a falling object. Again the lesion may be produced by direct force applied to the sternal end of the clavicle. Occasionally it is seen in debilitated individuals in whom there is gradual relaxation of the sternoclavicular and the costoclavicular ligaments allowing subluxation or dislocation of the proximal end of the clavicle.

Traumatic dislocations are associated with disruption of the sternoclavicular ligaments the interarticular disk retaining its attachment to the clavicle and following it in the dislocated position. In forceful downward displacements of the shoulder the proximal end of the clavicle is forced downward and backward until it impinges upon the first rib which acts as a fulcrum. If the force continues the first rib levers the inner end of the clavicle upward and forward tearing the sternoclavicular ligaments. This is the most common mechanism of production of sternoclavicular dislocation.

The final position of the end of the clavi-

cle depends upon the direction of the dislocating force. It may be displaced upward downward forward or backward. The most common displacement is forward rarely backward. Backward displacement results from direct violence applied to the sternal end of the clavicle or indirect forces driving the shoulder forward and inward. The writer has seen one instance of backward dislocation resulting from a direct blow with the butt of a rifle on the proximal end of the clavicle which was driven into a retrosternal position causing pressure on the trachea and the great vessels and producing *dyspnea and cyanosis*.

Like the acromioclavicular joint, the stability of the sternoclavicular joint depends upon its ligamentous structures and not on its bony configuration. Reduction of the deformity is accomplished readily although *maintenance of reduction is extremely difficult* (Fig 185).

TREATMENT

Recent Dislocation. Reduction is readily accomplished by the same maneuvers employed to reduce fractures of the clavicle. Replacement of the sternal end of the clavicle occurs when the shoulder girdle is pulled upward outward and backward. Maintenance of reduction is achieved by methods employed for fractures of the clavicle.

the first rib, keeping within the periosteal tube and close to the bone to avoid injuring the soft tissue structures posteriorly. Into the eye of the needle is threaded a piece of silk with a strip of fascia lata (from eight to ten inches long and one half inch wide) attached to the other end. The fascia is pulled around the rib and, by the same maneuver around the clavicle.

The process is repeated so that two loops of a continuous strip of fascia are passed around the clavicle and the first rib. The ends are then pulled tight and sutured to one another by interrupted silk or cotton sutures. Medial to the first fascial loop a second strip of fascia is passed around the first rib in the same manner as above. It is passed through a vertical drill hole in the clavicle. The ends are drawn taut and sutured to one another.

Postoperative Treatment. A Velpeau bandage is used to protect the shoulder for three or four weeks. A regimen of physical therapy and exercises is then begun. Optimum restoration of function is attained in from eight to ten weeks.

Arthrodesis of the sternoclavicular joint never should be done, because marked restriction of elevation of the arm results. Disability resulting from failure of reconstructive methods or arthritis may be relieved in a large measure by excision of one or one and one half inches of the proximal end of the clavicle.

DISLOCATIONS OF ACROMIOCLAVICULAR JOINT

Being located at the tip of the shoulder the acromioclavicular joint is vulnerable to such repeated trauma as direct forces applied to the top of the shoulder from above (falling objects) or falls on the point of the shoulder. Being a hinge joint, the acromioclavicular joint depends upon strong ligaments for stability. The articular surface of the clavicle faces downward outward and backward while that of the acromion looks upward inward and forward. Little stability is provided by the articular capsule

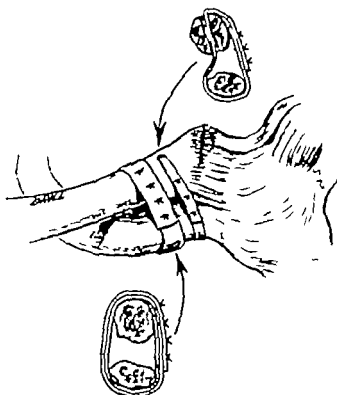


FIG 188 Reconstruction of sternoclavicular ligaments. Operation of Speed and Smith.

and the acromioclavicular ligament, the joint depending entirely upon the coracoclavicular ligaments (conoid and trapezoid ligaments) for its integrity. Depending upon the degree of disruption of the ligamentous apparatus of this joint, two lesions may occur (1) subluxation and (2) complete dislocation.

SUBLUXATION OF ACROMIOCLAVICULAR JOINT

Minor injuries may result in tearing of the articular capsule and the acromioclavicular ligament. Both the conoid and the trapezoid ligaments remain intact. Usually there is some drooping of the shoulder and slight anterior displacement of the acromion (Fig 191). Urist recorded that if in the anteroposterior roentgenograms there is widening of the joint space, the clavicle is displaced posteriorly. This is true even when the superior and the inferior surfaces of the clavicle and the acromion appear to be in normal anatomic relationship. The

instances the lesion is perfectly compatible with good painless function of the shoulder girdle. However, in other instances there is pain and restriction of elevation and abduction of the arm and the appearance of the deformity may be objectionable. In these cases operative procedures have been designed to effect a cure. Conservative closed methods are valueless. Most open methods attempt to anchor the end of the clavicle to the first rib (occasionally to the second rib), the manubrium or to both with fascia lata.

All procedures are difficult and tedious. Profuse bleeding may add to the difficulties of the operation. Not infrequently the joint is so disorganized that the interarticular disk must be removed, and secondary degenerative changes following repair are unavoidable. In the face of such difficulties some operators prefer to excise subperiosteally one to two inches of the sternal end of the clavicle. Such a procedure is compatible with good painless function and decreases the period of recovery without immobilization of the extremity.

Speed's operation for reconstruction of the sternoclavicular ligaments has given the

writer satisfactory results. Essentially, it anchors the mesial end of the clavicle to the first rib by two strips of fascia lata (Fig. 188). Bankart's operation is also useful to repair chronic dislocations of the sternoclavicular joint (Fig. 189).

Operative Technic (Speed) A straight skin incision from two and one half to three inches long is made along the inferior border of the proximal end of the clavicle beginning just lateral to the midline of the manubrium. Approximately two inches of the mesial end of the clavicle is exposed by stripping it subperiosteally. (At this point the writer investigates the sternoclavicular joint. If the meniscus is found to be disrupted or degenerated, it is excised.)

Part of the pectoralis major muscle fibers arising from the clavicle, the sternum and the first and the second costal cartilages are divided and reflected downward and outward bringing, into view the first and the second costal cartilages. The periosteum on the first rib is cut horizontally and the rib and the costal cartilage are exposed subperiosteally for about two inches.

An aneurysm needle is then passed around

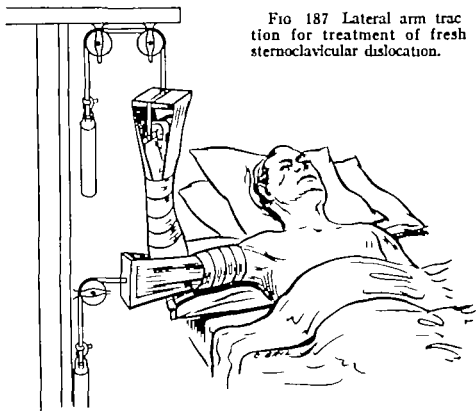


FIG. 187 Lateral arm traction for treatment of fresh sternoclavicular dislocation.

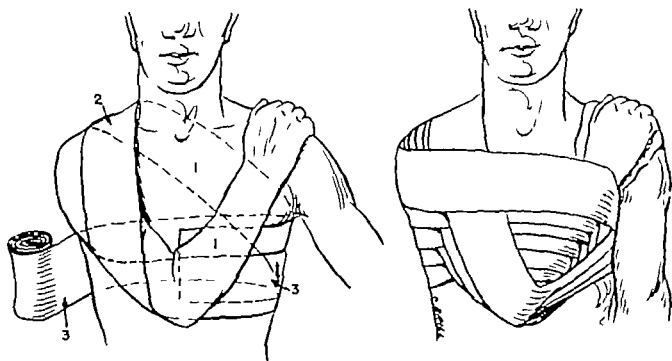


FIG 190 Velpeau bandage.

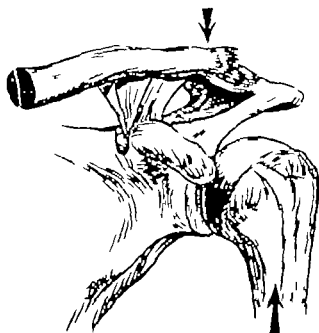


FIG 191 Subluxation of acromioclavicular joint.



FIG 192 Dislocation of acromioclavicular joint.

acromion is widely separated from the clavicle (Fig 192). Occasionally a segment of the outer end of the clavicle may be fractured and follows the acromion downward. As in subluxation of this joint the displacement is reduced readily by elevating the scapula by pushing the elbow upward and

backward, at the same time pressing the clavicle downward. However, release of all support results in recurrence of the deformity.

Diagnosis of the lesion by clinical examination is not difficult. Inspection discloses a droop of the shoulder downward

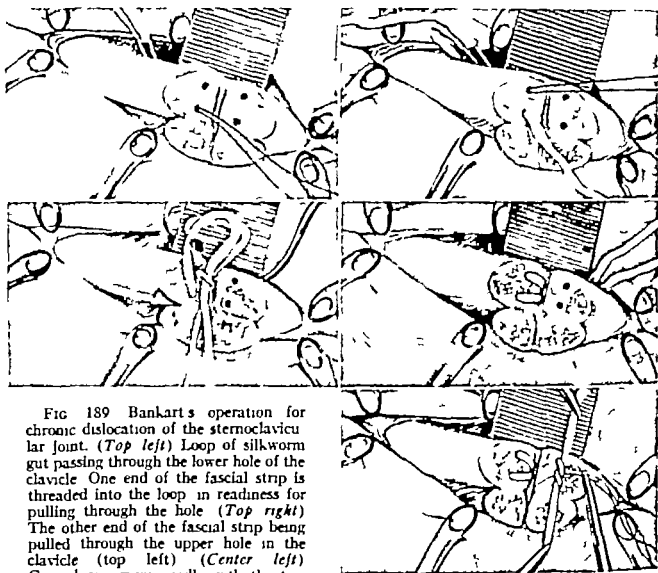


FIG 189 Bankart's operation for chronic dislocation of the sternoclavicular joint. (Top left) Loop of silk worm gut passing through the lower hole of the clavicle. One end of the fascial strip is threaded into the loop in readiness for pulling through the hole. (Top right) The other end of the fascial strip being pulled through the upper hole in the clavicle. (top left) (Center left) Curved aneurysm needle with the two ends of the fascial strip threaded through the eye prior to being pulled through. (center right) (Center right) The fascial strip pulled through to the back of the joint. (Bottom) The ends of the fascial strip have been brought through the holes in the sternum and tied and are held tight by an assistant while the surgeon sews the joint firmly with linen thread sutures. (Brit. J. Surg. 26 321 322)

same observer further noted that if the distal end of the clavicle was free floating (ballotement) after 3 weeks of conservative treatment this form of therapy would not effect a cure. The prime factors responsible for failure were interposition of the torn meniscus remnants of the torn acromioclavicular ligaments and detached portions of articular cartilage.

In subluxation of the joint, anteroposterior roentgenograms of both shoulders made with the patient standing and the arms at the side disclose that the interval between the clavicle and the coracoid process is the same on both sides. Pain may not

be severe but tenderness always can be elicited by pressure over the joint. Although the deformity may be slight in most instances a definite prominence of the clavicle can be demonstrated. The displacement is reduced readily by pressing the outer end of the clavicle downward while the elbow is pushed upward and backward.

COMPLETE DISLOCATION OF ACROMIOCLAVICULAR JOINT

This lesion is the result of violent trauma producing complete rupture of the coracoclavicular and the acromioclavicular ligaments. As the shoulder drops forward, the



FIG 194 Complete dislocation of the acromioclavicular joint. The interval between the clavicle and the coracoid process has been greatly increased.

clavicle, down the arm anteriorly, around the elbow, up the arm posteriorly and across the outer third of the clavicle anteriorly (Fig 195). Four or 5 such strips are superimposed on each other. This strapping is reinforced every 4 or 5 days by adding another strip.

Immobilization is maintained for from 3 to 5 weeks. Active motion within the limits of the dressing should be carried out at the shoulder, the elbow and the fingers. Free use of the arm is permitted after all fixation is removed. Physical therapy at this time in the form of radiant heat and gentle massage adds to the comfort of the patient.

Complete Dislocation of Acromioclavicular Joint (Acute) Due to total disruption of the coracoclavicular ligaments conservative measures have been very unsatisfactory on the whole. However many workers advocate such methods as Stimson's adhesive dressing (or various modifications of it), the abduction plaster jacket and numerous braces, splints and harnesses to maintain reduction until healing is complete. In the hands of many including the writer these measures have proved to be inadequate.

The view that complete dislocations are treated best by operative intervention is

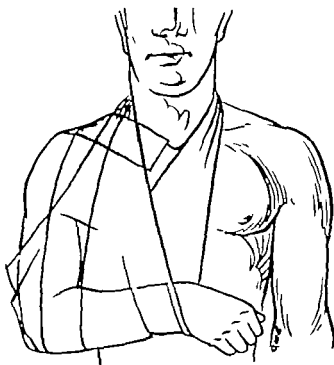


FIG 195 Stimson adhesive dressing for subluxation of the acromioclavicular joint.

becoming increasingly popular, and several procedures have been devised to restore the integrity of the acromioclavicular joint.

TRANSFIXION OF THE JOINT WITH WIRES This is an excellent method in acute complete dislocations. Done blindly or even with fluoroscopic control, the procedure is not without danger. Unpleasant complications are avoided if it is done under direct vision after exposure of the joint. Many complete dislocations, even after adequate re-establishment of the ligamentous structures are followed by arthritis of the joint, which may be responsible for much pain and disability. Degeneration of a traumatized meniscus (if present) is in a large measure the cause of the complication. Exposure of the joint permits visualization and excision of the meniscus and the removal of other tissue debris, thereby reducing the chance of such undesirable sequela.

Technic A straight skin incision is made starting on top of the clavicle $\frac{1}{8}$ inch to the inner side of the acromioclavicular joint

and forward and a prominent outer end of the clavicle (Fig 193) Pressure over the joint elicits marked tenderness A groove is palpable between the outer end of the clavicle and the acromion which is not palpable on the unaffected side If the acromion is steadied with one hand, the outer end of the clavicle can be displaced with ease up and down forward and backward, with the index finger and the thumb of the opposite hand

Roentgenographic examination reveals upward displacement of the clavicle and greater distance between the clavicle and the coracoid process on the injured than on the uninjured side (Fig 194)

TREATMENT OF RECENT LESIONS OF ACROMIOCLAVICULAR JOINT

Reduction of the displacement is accomplished easily but maintenance of reduction is extremely difficult.

Subluxation in which the coracoclavicu-

lar ligaments are intact is treated adequately by Stimson's adhesive dressing

STIMSON ADHESIVE DRESSING The extremity is suspended by a collar-cuff sling. One piece of felt is placed over the outer end of the clavicle, another is placed below the elbow, covering the proximal third of the ulna, and a pad of cotton is placed in the axilla. An adhesive strapping is then applied, so that the clavicle is pressed downward firmly, and the elbow is pushed upward This elevates the scapula and brings the acromion up to the clavicle Reduction must be maintained manually, while the dressing is being applied, by pushing upward on the humerus at the elbow and downward on the clavicle Skin irritation is prevented by applying a coating of benzoin tincture to the skin before applying the adhesive strips A second strip of adhesive 3 inches wide and 4 feet long starts posteriorly at the level of the waist and is brought upward over the outer end of the



FIG 193 Anterolateral and posterior views of a dislocated acromioclavicular joint, 1 month's duration. Observe the prominent outer end of the clavicle and drooping of the shoulder

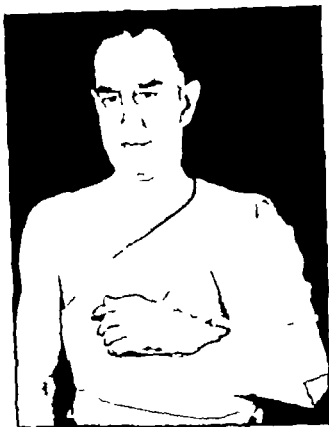


FIG 198 Plaster-of Paris shoulder swathe used instead of a Velpeau dressing to immobilize the arm



FIG 199 The outer end of the clavicle has been resected for an old acromioclavicular dislocation.

locations. However, it is now also widely used in fresh dislocations and the results are extremely satisfactory. As has been noted by many observers, the shoulder girdle, following excision of the outer end of the clavicle, does not regain complete function. The great majority of the patients complain of some weakness, a sense of instability and fatigue following strenuous activity. Nevertheless, normal range of painless motion is restored, the deformity is minimized and the convalescence is greatly shortened. It is particularly justified in individuals who are not employed in laborious occupations.

Technic. The distal end of the clavicle and the acromioclavicular joint are exposed by a curved incision. First approximately 1 to 1½ inches of the outer end of the clavicle is stripped subperiosteally of all soft tissue and then excised with a Gigli saw or bone-cutting forceps. The meniscus and other remnants of the acromioclavicular ligaments and the capsule are removed. The

remaining periosteum is utilized to cover the exposed raw surface of the end of the clavicle (Fig 199).

Postoperative Management. Complete rest of the extremity is desirable for about a week, during which time the shoulder is protected by a plaster-of-Paris shoulder swathe or by a Velpeau dressing. At the end of this period, soft tissue healing has progressed sufficiently to allow painless motion at the shoulder girdle. The patient is now permitted free use of the arm and progressive muscular exercises are begun. This phase of the treatment should aim for full restoration of muscle tone and power to stabilize the shoulder. Optimum restoration of function is attained in from 4 to 6 weeks.

RECONSTRUCTION OF CORACOCALVICULAR LIGAMENTS (using part of the conjoined tendon of the short head of the biceps and the coracobrachialis muscles). Several operations have been conceived to re-establish

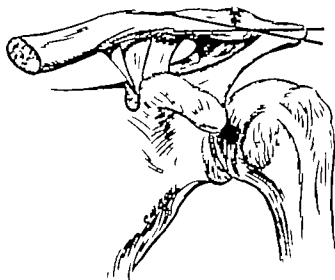


FIG 196 Transfixion of acromioclavicular joint by two threaded wires

and continues laterally to the tip of the acromion. The incision is deepened to the bone and the soft tissues are reflected by sharp dissection from the top of the acromion and the acromioclavicular joint. All remnants of capsular and acromioclavicular ligamentous tissue which may have dropped into the joint are removed together with the meniscus. Under direct vision while the clavicle is being held firmly in its normal anatomic position two stout Kirschner

wires are passed through the acromion across the joint space into the clavicle.

The wires penetrate the distal end of the clavicle for approximately 1 or 1½ inches. Their ends are then turned up and cut close to the edge of the acromion but left long enough to facilitate their withdrawal. The superior portion of the capsule and the acromioclavicular ligament is repaired as much as possible, and the wound is closed, leaving the ends of the wire beneath the skin.

Postoperative immobilization is accomplished best by a Velpeau dressing or a plaster swathe (Fig 198), which is worn for 4 weeks after which the arm is carried in a sling for 2 additional weeks. The wires may be removed at the end of 6 or 8 weeks at which time the patient is permitted free use of the extremity.

Instead of Kirschner wires, threaded steel pins may be utilized to transfix the joint. These have less tendency to migrate than the unthreaded pins or wires (Figs 196 and 197).

INCISION OF OUTER END OF THE CLAVICLE. This method was designed originally by Mumford and Gurd independently for treatment of chronic acromioclavicular dis-



FIG 197 (Left) Rupture of acromioclavicular joint (Right) The dislocation has been reduced and the acromioclavicular joint has been transfixied by two threaded wires the ends of which were cut off under the skin

observed that the tendinous origin of the conjoined tendon is rather broad and about $3\frac{1}{2}$ or 4 inches long. This tendinous segment is split in half longitudinally for its entire length. The distal end of the outer half of the tendon is divided transversely, while its proximal end remains attached to the coracoid process.

The next step comprises exposure and reduction of the dislocation. Through the upper arm of the incision the soft tissues are stripped by sharp dissection from the superior surface of the acromion and the outer end of the clavicle. All tissue debris, including the meniscus and the torn ligamentous and capsular tissues, are removed from the joint cavity. By firm pressure downward and forward on the clavicle the dislocation is reduced and maintained by passing two Kirschner wires or threaded wires through the acromion across the joint space and into the clavicle under direct vision. The acromioclavicular ligament is sutured in the best manner possible.

Next, that portion of the clavicle immediately above the coracoid process is stripped subperiosteally. The reflected portion of the conjoined tendon is passed around the clavicle subperiosteally and sutured to itself. The enveloping tube of periosteum is closed and sutured to the tendon within. The arm is immobilized in a plaster-of-paris shoulder swathe for 6 weeks. Graduated exercises and physical therapy follow and the wires may be removed at the end of 8 or 10 weeks. Maximum restoration of function is attained in from 12 to 16 weeks.

The writer has found this operation to be superior to other procedures employing strips of fascia lata and performs it chiefly for chronic rather than fresh dislocations. Calcification of fascia utilized in the reconstruction of the coracoclavicular ligaments is a frequent complication. To date this has not occurred in any of the cases in which the conjoined tendon was used. However it is feasible that calcification may occur. One must appreciate that calcification of tissues

in the coracoclavicular interval is a common complication of complete dislocation, even without surgical intervention. The subsequent synostosis limits elevation and abduction of the arm and is responsible for considerable pain and stiffness in the shoulder.

TRANSFIXION OF THE CORACOID PROCESS AND THE CLAVICLE WITH A SCREW. This method has been popularized by B. Bosworth, Vere-Hodge and Watson Jones. Essentially, it consists of maintaining reduction by means of a lag screw passed through the clavicle into the coracoid process. It has failed to meet the expectations of many of its supporters because the procedure is comparable with arthrosis of the acromioclavicular joint. Motion at this joint is necessary for complete elevation above the horizontal. This is accomplished by rotation of the clavicle in its long axis (Inman, Saunders and Abbott). Coracoclavicular transfixion eliminates all acromioclavicular motion thereby causing marked impairment of elevation of the arm.

TREATMENT OF OLD ACROMIOCLAVICULAR DISLOCATIONS

Not all old lesions demand surgical interference. In some individuals the dislocation causes no impairment of normal activity. In others, there is a pronounced disability characterized by a reduction of shoulder movements and by pain.

Excision of the outer end of the clavicle, as previously described, is applicable to all age groups and gives uniformly good results. It is especially indicated in individuals past middle life and in the aged.

In young athletic individuals and in those employed in strenuous occupations repair of the coracoclavicular ligaments is preferred. Repair is achieved either by using the conjoined tendon of the short head of the biceps and the coracobrachialis muscles, as described above, or fascia lata as described by Henry and by Bosworth or by the method of Bunnell (Fig. 201).

Restoration of normal anatomic relations

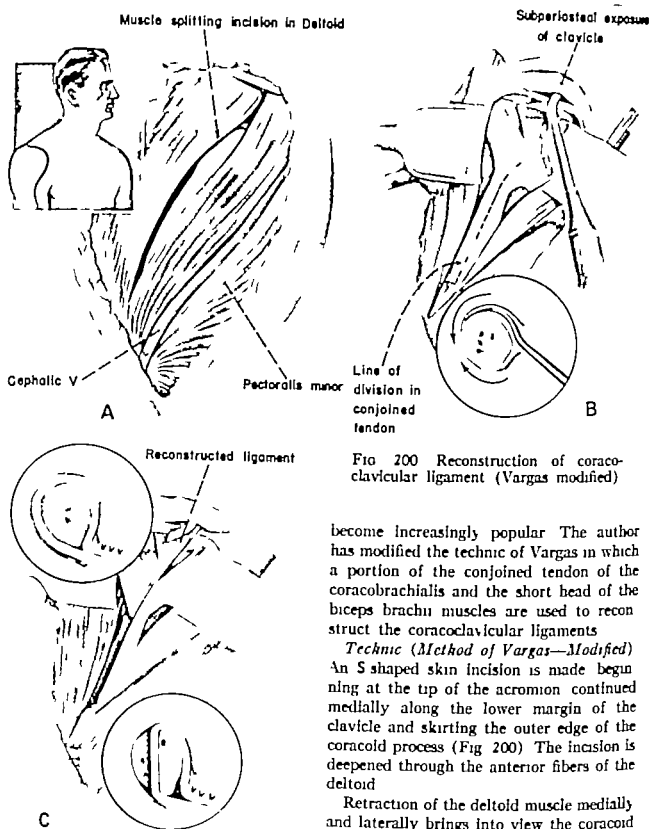


FIG 200 Reconstruction of coracoclavicular ligament (Vargas modified)

become increasingly popular. The author has modified the technic of Vargas in which a portion of the conjoined tendon of the coracobrachialis and the short head of the biceps brachii muscles are used to reconstruct the coracoclavicular ligaments.

Technic (Method of Vargas—Modified)

An S shaped skin incision is made beginning at the tip of the acromion, continued medially along the lower margin of the clavicle and skirting the outer edge of the coracoid process (Fig 200). The incision is deepened through the anterior fibers of the deltoid.

Retraction of the deltoid muscle medially and laterally brings into view the coracoid process and the tendinous origin of the conjoined tendon of the short head of the biceps and the coracobrachialis muscles. The interval between the deltoid fibers is developed proximally as far as the inferior border of the clavicle.

At this stage of the operation, it will be

the integrity of the ligamentous apparatus of the acromioclavicular joint. In the main, strips of fascia lata are utilized to reconstruct the disrupted ligaments. The operation devised by Bunnell and by Henry have

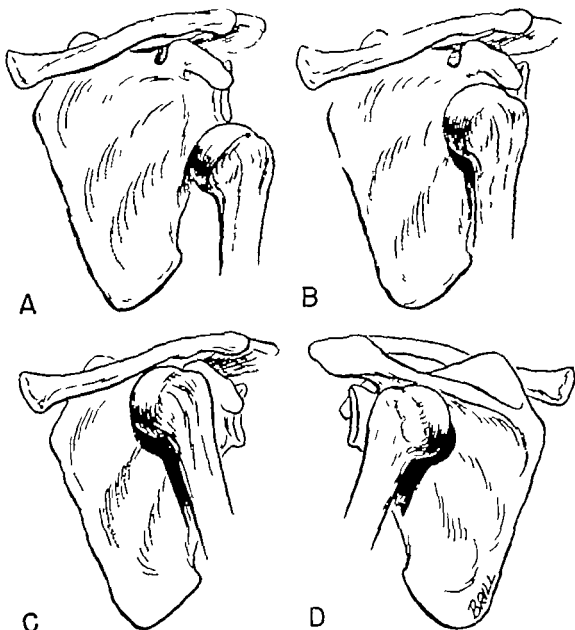


FIG. 202 Four types of dislocation of the scapulohumeral joint. (A) Subglenoid dislocation (rare type) (B) Subcoracoid dislocation (most common type) (C) Subclavicular dislocation (rare type) (D) Subspinous dislocation (rare type)

ments are relatively lax, thereby allowing a wide range of motion. As previously noted the integrity of this joint depends essentially upon the surrounding strata of muscles and atmospheric pressure. Moreover its unprotected position renders the joint extremely vulnerable to trauma.

Functionally the glenohumeral joint possesses a wide range of movements, made possible not only by its bony configuration but also by a delicate, intricate system of neuromuscular balance and co-ordination. If disturbed by unguarded sudden motions or violent injury dislocation or fracture of

the upper end of the humerus may result.

Types of Dislocations. Clinically four types of dislocation are encountered depending upon the position of the humeral head in relation to the glenoid cavity. The head may lie below the glenoid cavity (subglenoid dislocation), beneath the coracoid process (subcoracoid dislocation), occasionally under the clavicle (subclavicular dislocation), and in rare instances the head occupies a position below the acromion or the spine of the scapula (posterior or subspinous dislocation). Subcoracoid is the most frequent type of dislocation. The sub-

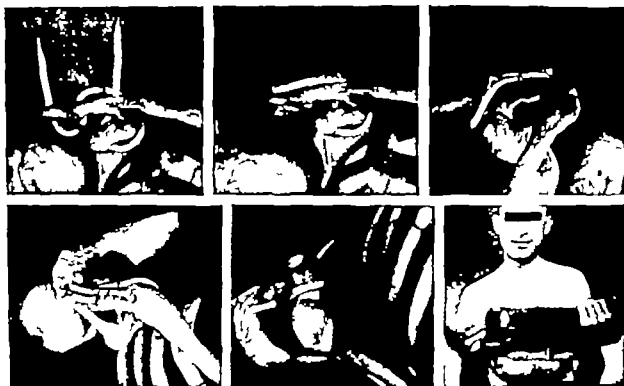


FIG 201 For dislocation of acromioclavicular joint. Method of placing 12 inch graft of fascia lata to reproduce conoid and trapezoid ligaments. (Top left) The anatomy of the conoid and trapezoid ligaments the positions of the three holes in the bone and the fascial graft (indicated by rubber tube) in place but not yet drawn up (Top center) Fascial graft (indicated by rubber tube) in place and sutured as seen from the front. (Top right) Posterior view (Bottom left) Superior view showing extra reinforcement of the joint capsule (Bottom center) Inferior view (Bottom right) Patient on whom this operation was performed 6 months previously. Although he is lifting a heavy anvil, no difference can be detected in his two acromioclavicular joints. The original anatomy is reconstructed and the function is normal (Surg. Gynec. & Obst. 46 563)

In the acromioclavicular joint does not ensure a painless freely moving shoulder girdle. Pain and disability may persist even after operative re-establishment of the torn ligaments. Degenerative changes of varying degrees in the joint are to be anticipated.

Arthrodesis of the acromioclavicular joint never should be done because of the subsequent restriction of elevation. Arthritis regardless of the degree or disruption of the acromioclavicular joint does not justify an arthrodesis. Excision of the outer end of the clavicle is the procedure preferred for such cases.

DISLOCATIONS OF THE SCAPULOHUMERAL JOINT

ACUTE DISLOCATIONS

General Considerations. Dislocation at the scapulohumeral joint is a frequent

lesion estimates indicating that from approximately 40 to 60 per cent of all dislocations occur at this joint. The lesion is encountered most frequently in the late teen years and after the age of 20 but they may occur at any age. Young athletic males are usually affected but the condition may occur in females. It is a rarity in children.

Several anatomic and functional features of the shoulder are responsible for the frequency of dislocation. Anatomically the bony architecture of the joint favors instability. Only a very small area of the large spherical humeral head articulates with the shallow glenoid cavity at any one time. The fibrous capsule is loose and redundant becoming taut only at the extremes of the arcs of motion. Roughly its surface area is twice that of the humeral head. Likewise the various reinforcing capsular liga-

glenoid form is next in frequency. It is reasonable to assume that many subcoracoid lesions were first subglenoid and by inward and forward movement of the humeral head or the drive of the dislocating force were converted into subcoracoid dislocations (Fig 202).

Luxatio erecta is the rarest form. The arm is fixed to the side of the head and points directly upward, while the forearm rests on the top of the head; the humeral head lies in a subglenoid position. The lesion is produced by hyperabduction of the arm while an axial force drives the head of the humerus directly downward through the capsule.

MECHANISM OF PRODUCTION OF DISLOCATION

Survey of the literature discloses many diversified concepts of the mechanisms of dislocation, and there is no general agreement among the earlier observers. This confusion stems in part from the difficulty in obtaining an accurate account of the movements and the position of the arm at the time of the injury, and in part from the lack of comprehension of the normal movement of the glenohumeral joint. Codman has given us an unequivocal mechanism of dislocation which is based on a scientific analysis of the normal functions of the shoulder joint. He noted that 'no matter in what degree of rotation you start to raise your arm and no matter in what plane you raise it the capacity for rotation will be less and less as the arm ascends until in complete elevation tuberosities and processes will be locked in a fixed position. This position was designated the pivotal position. In order to reach the pivotal position in the sagittal plane the arm must be rotated internally, and in the coronal plane rotated externally.

If during elevation of the arm in the sagittal plane internal rotation is prevented the humerus will impinge upon the acromion which acting as a fulcrum will lever the head of the humerus out of the glenoid cavity or produce a break in the bone. The

same is true if external rotation of the humerus is prevented when the arm is being elevated in the coronal plane. Hyperabduction of the arm beyond the limits of the pivotal position will also result in dislocation or fracture of the humerus (Fig 203). It is apparent that the position of safety is the pivotal position. During forward or lateral falls, the ascending arm instinctively rotates in the right axis to reach this position. However, if rotation in the correct plane is prevented, or if the falls are violent and sudden, catching the muscles off guard in a relaxed state before they can rotate the humerus into the position of safety, disruption of the glenohumeral joint is inevitable.

One can demonstrate readily on a subject that the scapulohumeral joint will lock in different positions, depending upon the degree of rotation of the humerus. This is true, regardless of the relation of the humerus to the scapula, whether it be in the anatomic position (the arm at the side with the palm facing forward), at the horizontal or in the pivotal position where no rotation is possible. If the limits of this locked position are exceeded the humerus obtains a fulcrum on the edge of the acromion, and dislocation or fracture of the humeral head ensues.

The mechanism of dislocation when the falling patient exerts force on the outstretched arm is the same as when leverage is applied on the arm with the body in a fixed position. Anterior or posterior dislocations will occur depending upon the direction of the disruptive forces. As a rule anterior dislocation occurs if hyperabduction is continued after the arm has reached the pivotal position or if rotation is prevented after the arm has ascended to a point above the horizontal. Posterior dislocation occurs when the arm is below the horizontal and is rotated internally beyond the limits of the locked position.

Analysis of the many different combinations of elevation and rotation of the humerus which are likely to produce dislocation indicates that clinically, the mechanism

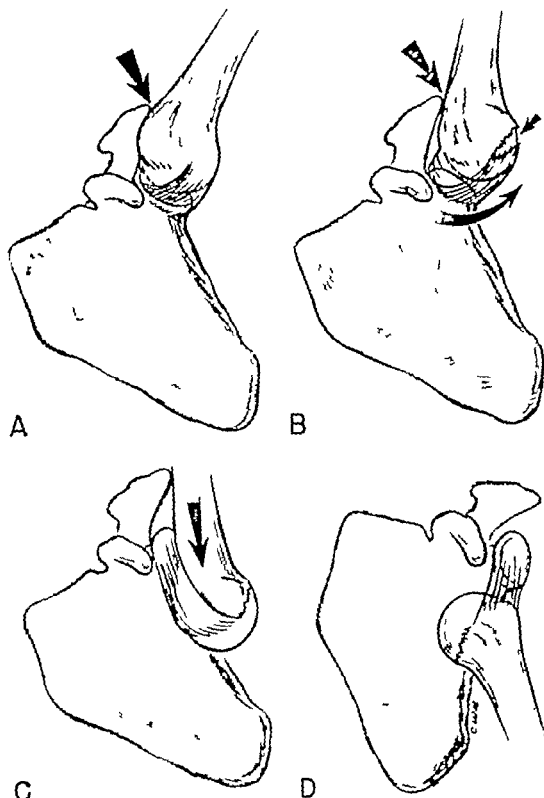


FIG 203 Mechanism of production of acute traumatic dislocation of the scapulohumeral joint by hyperabduction of the arm (A) Arm in the pivotal or safety position. (B) Humerus is forced into hyperabduction impinging against the acromion which acts as a fulcrum and levers the head of the humerus out of the glenoid cavity note the tearing away of the inferior portion of the capsule from the neck of the humerus. (C) Head leaves glenoid fossa through rent in capsule stretching the rotator cuff across the glenoid cavity (D) Arm at the side with head of humerus assuming first a subglenoid position.

This is the most common mechanism of anterior dislocations (Fig 204)

Adams lists five types of injuries which may be responsible for initial dislocation

- 1 Fall on the abducted arm (without hyperabduction)
- 2 Direct blows on the head of the humerus from behind
- 3 Hyperextension of the abducted arm
- 4 Excessive external rotation in abduction
- 5 Hyperabduction (causing inferior dislocation)

Critical analysis of the above types make it clear that all of them can be explained by Codman's postulates of dislocation

PATHOLOGY OF ACUTE ANTERIOR DISLOCATION

Much has been recorded on the pathology of acute anterior dislocation. Many of the proposed concepts disclose total disagreement thereby giving rise to considerable confusion. This occurs because hitherto many of the postulates of dislocation were based on pure conjecture rather than actual visualization of the affected tissues at the time of the injury. According to most observers, avulsion of the labrum glenoidale, the fibrous capsule and the glenohumeral ligaments from the antero-inferior aspect of the bony rim of the glenoid fossa represents the most frequent pathologic lesion. Many observers (Ivan Palmar, Adams, King, Gallie and Eyre Brook) are of the opinion that a compression fracture of the posterior aspect of the head of the humerus may occur at the time of the first dislocation and recurrences of the fracture are likely.

Fracture of the bony rim of the glenoid fossa has been described by many investigators. Bankart emphasizes that there are two distinct types of traumatic dislocations. He contends that the ordinary traumatic lesion is an inferior dislocation, the head of the humerus passing through a rent in the capsule. It is Bankart's belief that this lesion heals readily and is not followed by

recurrences. The second type is anterior dislocation associated with detachment of the labrum and the capsule from the anterior margin of the glenoid cavity. Frequently, this lesion is followed by recurrences, because the detached labrum fails to reattach itself to the glenoid rim.

It is obvious that clarification of the pathology of acute dislocation can be attained only by exploration of the shoulder joint at the time of injury. Such exploration was performed in 18 fresh anterior dislocations before reduction. No cases complicated by fracture of the humeral head are included in this study. Ages of patients ranged from 18 to 64 years, 6 were over 45, and the remainder were under 35 years of age. All patients were male.

In all instances roentgenographic examination disclosed a subglenoid or subcoracoid dislocation. Care was taken to hold the humeral head in its abnormal position until it was visualized.

Pathologic Observations

POSITION OF HUMERAL HEAD IN RELATION TO THE GLENOID CAVITY In two cases the humeral head was found in a subglenoid position, resting immediately below the inferior rim of the glenoid cavity. In 16 cases it occupied a subcoracoid position.

POSITION OF HUMERAL HEAD IN RELATION TO CAPSULE

Head Outside of Capsule In six instances the heads were outside of the fibrous capsules. In four cases it was noted that the inferior capsule had been torn away from the neck of the humerus, allowing the head to escape from within the capsule. Two of the four shoulders demonstrated the rent in the capsule to be continuous with a tear in the substance of the subscapularis tendon where it inserted into the lesser tubercle. Inspection of the interior of the joint cavity in all four shoulders disclosed the labrum and the glenohumeral ligaments to be intact.

The remaining two humeral heads revealed massive cuff tears, both heads being outside the joint cavity lying beneath the coracoid process. It was significant that both

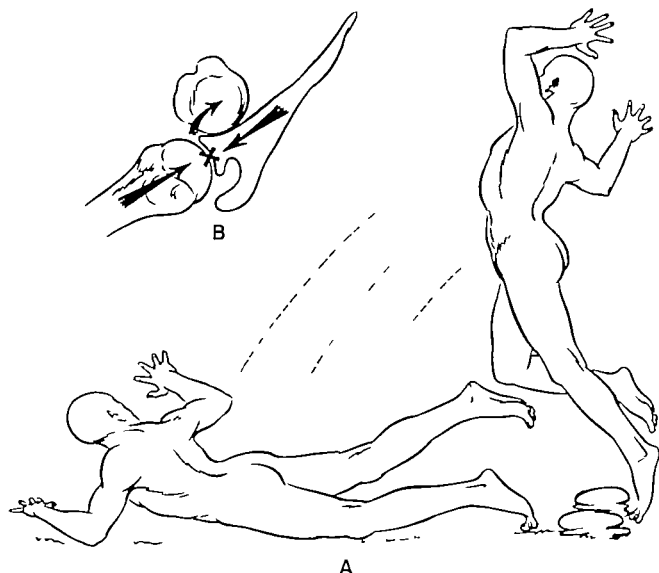


FIG 204 (A) Dislocation caused by fall on outstretched arm producing an impacting force against the glenoid cavity (B) Resultant force is created which acts through the plane of glenohumeral joint and allows head to dislocate anteriorly on the neck of the scapula in a subcoracoid position

of production is not constant. Whether some of these mechanisms are more likely to initiate recurrent dislocation than others is discussed under Pathology of Recurrent Dislocation.

Other factors which play a significant role in the mechanism of dislocation are (1) the action of the adductor muscles and (2) the impacting or telescoping force applied to the glenoid cavity through the bony axis of the humerus during a fall on the outstretched arm or the partially abducted elbow. Codman holds that the contracted adductor muscles (pectoralis major and

latissimus dorsi) do not create a fulcrum but promote dislocation by acting as internal rotators thereby preventing external rotation of the humerus when the arm is ascending in the coronal plane.

Falls on the outstretched arm or on the partially abducted elbow create an impacting or telescoping force which is resisted by the glenoid cavity. A resultant force ensues which acts through the plane of the glenohumeral joint. If the force is of sufficient severity the humeral head is forced forward on the anterior surface of the neck of the scapula usually in a subcoracoid position.

of bone devoid of soft tissue attachments, lay within the joint cavity. In another shoulder, a piece of the rim of the glenoid was found with a segment of the labrum still adherent to it (Fig. 205). Six shoulders disclosed no abnormalities of the labra, but all the glenohumeral ligaments and the anterior portions of the capsules suffered extensive tearing and attenuation.

It was interesting to note in all these specimens that the head came to rest finally in the subscapularis recesses. Because of the laxity of the capsule and the glenohumeral ligaments one can apprehend how the boundaries of the above recesses can stretch or tear readily if necessary, to admit the humeral head. In no instance, however, was the head found out of the confines of the fibrous capsule. This is explainable by the topographic anatomy of this region. As noted in Chapter 3, in the greater majority of cases the upper two-thirds of the anterior portion of the fibrous capsule does not insert directly into the labrum and the rim of the glenoid cavity but is continued mesially as far as the subcoracoid region where it is reflexed along the anterior aspect of the neck of the scapula to the capsular margin of the labrum.

This outpouching of the fibrous capsule forms the subscapularis recesses, either one or two depending upon the presence or the absence of a middle glenohumeral ligament (Fig. 227).

From this study it is apparent that most dislocations are intracapsular, that the head comes to rest in the bursal recesses and not out of the joint capsule.

No defects comparable with fresh compression fractures or contusions were demonstrable in the posterior aspect of the humeral heads.

Nicola's comprehensive study on a similar series of cases adds another important observation to those recorded above. He noted that in 22 cases the capsule was torn from the neck of the scapula and in 5 cases it was torn from the humerus. In the former group the lesion was the result of abduction



FIG. 205 Fragments of torn labrum and pieces of glenoid rim removed from within joint cavity in a case of acute dislocation of the glenohumeral joint.

plus an impacting force acting against the glenoid cavity. In the latter group simple hyperabduction of the arm was responsible for stripping the capsule from the neck of the humerus.

From the afore mentioned investigation in initial dislocations without fractures the pathologic observations may be summarized as follows:

- 1 Stripping the capsule from the inferior aspect of the humeral head
- 2 Disruption of the glenohumeral ligaments and attenuation of the boundaries of the subscapularis recesses
- 3 Shearing the labrum glenoidale from the anterior glenoid rim
- 4 Fracture or maceration of the labrum glenoidale
- 5 No involvement of the labrum
- 6 Stripping labrum capsule and periosteum from the anterior surface of the neck of the scapula
- 7 The humeral head lies extracapsular when it is forced through a rent in the capsule made by stripping the capsule from the humerus or when it is driven through a tear in the cuff
- 8 The humeral head is intracapsular when it lies within the confines of the subscapularis recesses
- 9 Varying degrees of tears of the rotator cuff involving chiefly the subscapularis and

these patients were over 60 years of age. In one the cuff tear involved the superior half of the subscapularis tendon, the supraspinatus and the infraspinatus tendons. In the other, in addition to avulsion of the supraspinatus and the infraspinatus portion of the cuff from the humeral head, a long longitudinal split was demonstrable in the interval between subscapularis and supraspinatus tendons. In both instances the biceps tendon was found outside of the groove stretched over the greater tuberosity. It was apparent that the heads were forced through the traumatic defects in the cuffs. In the first case the avulsed cuff lay in front of the glenoid cavity like a curtain. It became obvious that closed reduction in this case would fail because of the interposition of the torn rotator cuff. Inspection of the interior of the joints fails to exhibit any abnormalities of the fibrous capsule, the labrum glenoidale or the glenoid rim that could not be explained as degenerative changes associated with usage and senescence.

Head Inside of Capsule. Twelve shoulder joints exhibited the humeral heads on the anterior aspect of the neck of the scapula but within the joint capsule. In all instances the subscapularis muscle was stretched severely as it passed over the displaced head; its tendon fibers were blanched and many showed tiny transverse superficial tears. Moreover the other rotators were all under tremendous tension as they were pulled tightly over the empty glenoid fossa. In all instances the supraspinatus muscle appeared to be under the greater strain. The infraspinatus and the *teres minor*, although under less strain, were angulated sharply over the posterior edge of the glenoid fossa.

Four cuffs disclosed a complete tear. Of these two involved the upper half of the subscapularis tendon and the supraspinatus tendons, one the supraspinatus and the infraspinatus tendons and one only the supraspinatus tendon. Although no obvious tears were discernible, all the remaining cuffs were stretched severely across the glenoid

cavity. In one cuff which revealed a tear, the biceps tendon was found outside of the intertubercular sulcus lying against the intact edge of the infraspinatus tendon.

In order to visualize adequately the glenoid fossa and the structures in its anterior aspect, the dislocation was first reduced. The subscapularis tendon and with its capsule was then divided 1 cm. from its insertion into the lesser tuberosity and was retracted medially. It was of interest and most significant to note the diversity of pathologic observations discernible in this region of the joint. It was obvious that all soft tissue elements were stretched to accommodate the head. By passing a probe along the anterior margin of the glenoid cavity beneath the articular edge of the labrum or along its capsular margin in no instance was it possible to find a direct communication between the interior of the joint and the exterior.

As a rule, the inferior and the middle glenohumeral ligaments elicited evidence of pronounced trauma, particularly the inferior ligament which was either completely torn from its labral attachment or markedly attenuated. Two inferior ligaments were split longitudinally. Several shoulders disclosed ruptures of the labral ends of the middle ligaments, allowing them to dangle freely within the joint cavities from their intact humeral attachments. Varying degrees of separation of the labra were noted. In six shoulders the labra were separated completely from the anterior portion of the glenoid rim, exposing the raw bony surface. However in all instances the reflexed capsule over the neck of the scapula was intact. Three of these 6 cases also demonstrated crushing and shredding of the labra, leading one to conclude that the structures were subjected to great shearing strains. Two cases disclosed stripping of the inferior ligament from its labral attachment together with the reflexed capsule from the neck of the scapula. In one instance with a detached labrum the bony rim of the glenoid cavity was fractured completely and the fragment

Pain and muscle spasm may preclude a diagnosis of loss of motor power in the deltoid muscle prior to reduction. At this time, the only significant evidence of circumflex nerve affection is the presence of a

patch of hypoesthesia in the sensory distribution of the nerve through its cutaneous branch, the upper lateral cutaneous nerve of the arm (Fig 207). As a rule, impaired skin sensation in the deltoid area returns

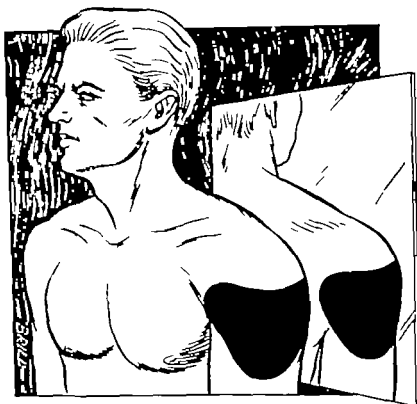


FIG 207 Area of hypoesthesia or sensory loss, following injury to circumflex nerve



FIG. 208 Area of impaired sensation following lesions of the musculocutaneous nerve

rapidly to normalcy after reduction. In most instances in which loss of motor power is manifest after reduction recovery eventually takes place. This, however, may require many weeks and even months. Only rarely does circumflex palsy become a permanent lesion.

MUSCULOCUTANEOUS NERVE INJURIES

Like the axillary nerve, the musculocutaneous nerve may be traumatized by the same abnormal mechanics of a dislocating head or it may be injured by direct pressure of the head which has been left in a dislocated position for a long period of time. Clinically, the diagnosis is made by paralysis of the biceps brachii, the coracobrachialis and the brachialis muscles and by reduced sensibility along the lateral border of the forearm (Fig 208). Injury to the nerve is

the supraspinatus region (tears may be transverse or longitudinal) Eight of the 16 shoulders exhibited macroscopic tears of some degree in the fibrotendinous cuff Six of the 8 affected cuffs disclosed a defect in

OTHER COMPLICATIONS EXCLUSIVE OF FRACTURE

Nerve Lesions

AXILLARY CIRCUMFLEX NERVE INJURIES. During dislocation, tremendous lateral pres-

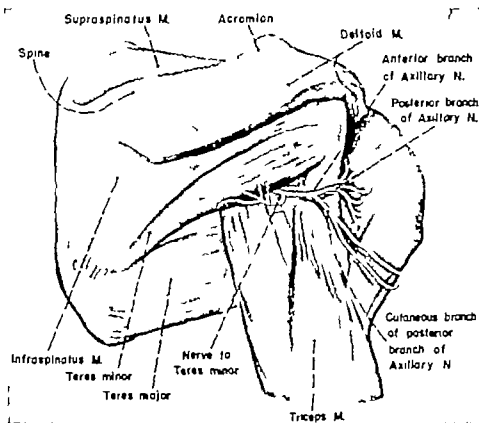


FIG 206 Course of axillary nerve as it leaves the quadrangular space The portion of the nerve proximal to the point of exit is the segment most apt to be injured in dislocations of the scapulohumeral joint.

the supraspinatus region In the light of this information it is reasonable to conclude that many of these cuffs sustained incomplete not demonstrable tears on the joint side of the cuff and that all sustained microscopic tears

10 Displacement of the biceps tendon from the bicipital sulcus

11 Fracture of the anterior long rim of the glenoid cavity

12 Severe stretching of the rotator muscles particularly the supraspinatus and the subscapularis muscles. This feature was constant in all the shoulders explored Its significance in the mind of the author has been underestimated in relation to development of recurrent lesions

sure may be exerted on the brachial plexus in the axilla by the abducting and rotating humeral head Although any of the nerves may be involved the axillary nerve is injured most frequently It has been estimated that approximately 25 per cent of all dislocations are accompanied by axillary nerve trauma. The course and the position of the nerve in relation to the path of a dislocating humeral head renders it extremely vulnerable (Fig 206) Fortunately stresses on the axillary nerve are rarely sufficient to cause complete ruptures but are often of sufficient severity to cause paralysis of the deltoid muscle In ruptures that have been explored the lesion occurred proximal to the exit of the nerve through the quadrangular space

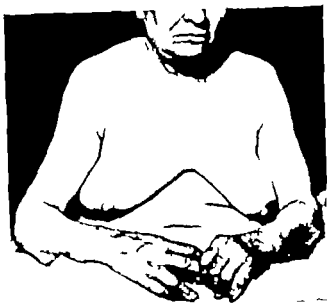


FIG 210 Subcoracoid dislocation of the right scapulothumeral joint. Normal rounded contour of the shoulder is lost the shoulder is flattened and the elbow projects from the side the arm appears lengthened when compared with the unaffected extremity



FIG 211 Subcoracoid dislocation. Observe small fragment of bone sheared from the inferior portion of the glenoid rim.

opportunity to recognize early concomitant lesions. The characteristic features are

1 History of injury is always present. Occasionally it is trivial but in most cases it is violent in character

2 Pain and disability ensue. Any attempt to move the arm actively or passively accentuates the pain

3 Inspection discloses that the arm is maintained in a fixed position of slight abduction (25° to 30°) the long axis of the humerus being directed upward and inward. The forearm is rotated internally and flexed. The arm appears to be lengthened. The distance from the acromion to the external epicondyle on the injured side is greater than that on the normal side

The patient tends to lean toward the affected side to allow the extremity to hang in a vertical position. The shoulder is flattened. This is more noticeable if the patient is observed from behind while the acromion process is prominent. The axillary fold is fuller and at a lower level than that on the opposite side. An abnormal prominence is discernible in the subcoracoid region (Fig 210)

4 Palpation of the area reveals a noticeable defect below the acromion and an absence of the globular mass made by the humeral head normally felt in this region. However the humeral head can be palpated in its abnormal subcoracoid position. Rotation of the arm discloses the humeral head moving with the humeral shaft.

5 ROENTGENOGRAPHIC EXAMINATION is an essential part of the examination because it not only definitely establishes the diagnosis but demonstrates concomitant fractures of the humeral shaft or tuberosities which if present will alter the form of treatment (Fig 211)

While conducting the examination one never should fail to note the presence or the absence of areas of decreased sensitivity of the skin supplied by the sensory branch of the circumflex nerve. One should examine the deltoid, the pectoralis major and the rotator muscles also the muscles of the arm and the forearm for motor power. Notation

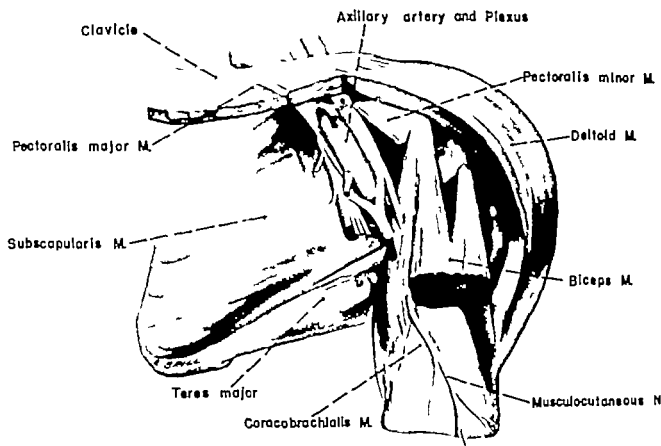


FIG 209 Course and vulnerable position of brachial plexus and musculocutaneous nerve which may be injured at the time of the dislocation or by pressure of the head of the humerus left in a dislocated position for a long period of time.

encountered only rarely. It occurs however next in frequency to injuries of the circumflex nerve.

Many other branches of the brachial plexus may be traumatized and inasmuch as the consequences of inadequately treated nerve lesions are so devastating it is mandatory that at least a cursory neurologic examination be done in all cases of acute dislocation of the shoulder before reduction (Fig 209). A second evaluation of the brachial plexus should be done after reduction. Only by such a procedure is one able to recognize nerve lesions early and determine definitely whether they complicated the dislocation or were the result of the reduction.

VASCULAR INJURIES Although rare in injuries to the axillary artery or vein have been reported. They may occur at the time of the injury or are produced by forceful maneuvers employed at reduction. The author has seen one instance of severance

of the axillary artery at the initial injury when the patient fell through the hatch of a ship and landed on the outstretched arm. Severance of the artery is made evident by obliteration of the radial pulse and a rapidly developing pulsating hematoma in the axilla or front of the shoulder. Rupture of the vein is followed by a more slowly developing hematoma. The extremity becomes markedly swollen and cyanotic. The vessels may be traumatized but not severed and aneurysm or arteriovenous fistula may develop.

FRACTURES OF THE HUMERUS

(These are considered under Fracture Dislocations.)

CLINICAL FEATURES OF ACUTE ANTERIOR DISLOCATION

Generally the diagnosis is so obvious that one often neglects to conduct a thorough examination of the part thereby forfeiting the

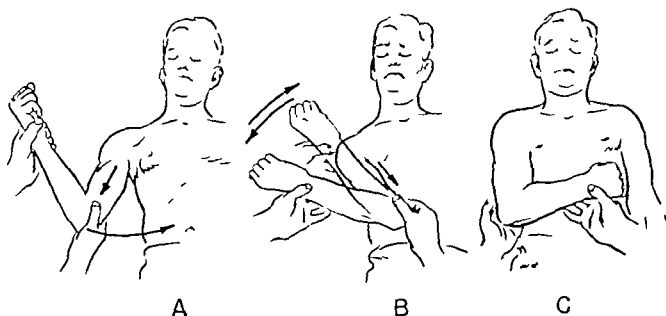


FIG 212 Reduction of dislocations of the scapulohumeral joint by steady traction on the humerus, first in the plane in which it lies in relation to the trunk (A) Traction is maintained while the arm is carried into a slightly adducted position this usually effects reduction Occasionally it may be necessary to rotate gently the arm externally and internally (B) After reduction, the arm is gently laid across the chest (C)

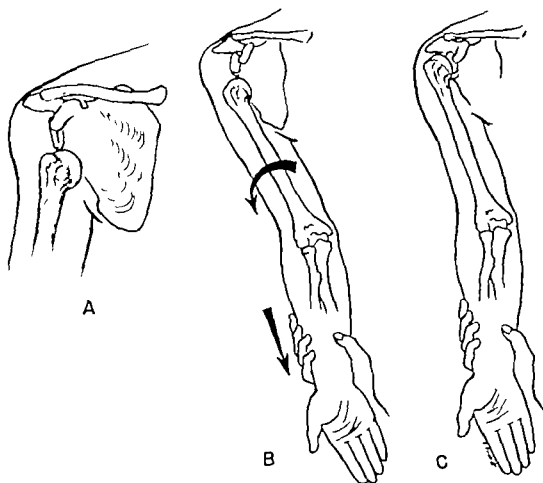


FIG 213 Reducing dislocations of the scapulohumeral joint as illustrated in Figure 212 except that traction is made with the arm extended instead of flexed.

of the vascular status of the arm never should be overlooked.

Treatment The objectives to be achieved in the management of an acute uncomplicated inferior or anterior dislocation of the shoulder are (1) reduction without further trauma to the capsular element, the musculotendinous cuff, the neurovascular structures and the bony components of the joint (2) maintenance of reduction to allow healing of the injured tissues and to prevent *redislocation* and (3) *restoration of normal function*. Of necessity, treatment may be modified in the presence of concomitant complications or to prevent undesirable sequelae.

One's choice of a method of reduction must be tempered by knowledge of the following clinical facts. Most uncomplicated recent dislocations are reduced readily with minimum force when pain and muscle spasm are eliminated provided that the method of reduction is executed properly. Difficulty in reduction is encountered only in the presence of an associated complication not recognizable by routine examination. Reduction should be performed as soon after the dislocation has occurred as is possible because at this time disalignment of the bony components of the joint is maintained primarily by muscle spasm unless as has been pointed out previously complicating lesions exist.

Later the displacement is fixed by infiltration and organization of the soft tissues. Elasticity of these structures is lost and now reduction can be effected only by more forceful maneuvers. In the light of the pathology of acute dislocations it becomes apparent that any method of reduction which employs abduction and rotatory movements is likely to add further trauma to disrupted tissues or to produce new injuries. Steady traction in the long axis of the humerus either in a neutral or slightly adducted position is the most essential maneuver of any method of reduction.

Choice of Anesthesia. Although many operators attempt reductions without a gen-

eral anesthesia, it is the author's opinion that a general anesthesia should be used in every instance. Complete muscular relaxation is essential to effect a reduction without force, and this cannot be obtained by the use of morphine or other opiates. Pentothal Sodium is a very useful agent for this purpose.

Traction on Adducted Humerus. This procedure will effect reduction in all uncomplicated early inferior and anterior dislocations with *minimum risk of added trauma*. It should take precedence over all other forms of reduction.

The patient lies on his back and with the elbow flexed to a right angle, the operator makes firm, steady traction in the line of the adducted humerus. Countertraction is provided by the patient's weight. If more is desired it may be attained by passing a folded sheet around the chest wall and pulling obliquely in the opposite direction. In many instances this maneuver alone will effect a reduction. Slight rotatory motions, while traction is being maintained may be added if necessary (Fig. 212). Some surgeons make traction with the arm extended at the elbow (Fig. 213). However flexion of the elbow to a right angle relaxes the biceps tendon and the neurovascular bundle thereby eliminating the risk of injury to these structures.

Kocher's Maneuver This procedure was designed primarily for subcoracoid dislocations. The various steps cause the humeral head to retrace its path into its normal anatomic position. When the operation is performed gently no harm ensues. However, if improperly executed severe damage to the brachial plexus and the humerus may result. At all times one must be cognizant of the fact that Kocher's manipulation is based on a series of rotatory movements of the humeral head also that great force can be exerted on the upper end of the humerus and the glenohumeral soft tissue elements by using the arm as a lever. Fractures of the humeral shaft, massive avulsions of the rotator cuff and injury to the circumflex

wrist with the left hand, steady traction is made in the line of the long axis of the humerus or preferably in slight adduction. Following this stretching phase the three Kocher steps are carried out (Fig 214).

EXTERNAL ROTATION Traction being maintained on the humerus, the forearm is moved outward gently and slowly to the limits of external rotation. The forearm now lies in a position of 70° to 80° external rotation. Such a maneuver stretches the subscapularis muscle and allows the head to move so it impinges against the anterior margin of the glenoid fossa.

FORWARD ADDUCTION Traction and external rotation being maintained the elbow is moved forward slowly and steadily to a point near the midline of the body. By this procedure the head is levered over the glenoid rim. As a rule reduction occurs during the execution of this step.

INTERNAL ROTATION While holding the elbow close to the midline, the arm is rotated internally by moving the forearm across the chest so that the hand falls over the opposite shoulder. This last step in the manipulation places the head in its normal anatomic position.

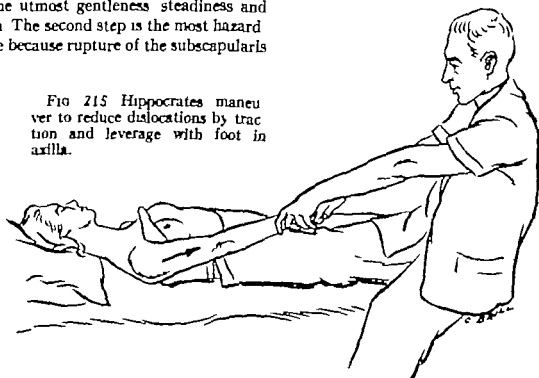
It cannot be overemphasized that all phases of this method must be exercised with the utmost gentleness, steadiness and caution. The second step is the most hazardous one because rupture of the subscapularis

tendon or fracture of the shaft may occur readily during its execution. Should the first attempt fail, another may be attempted, but a third attempt is contraindicated, because some unrecognized complication undoubtedly exists, and surgical intervention is now justifiable as a safer approach to the problem.

Traction and Leverage with Foot in the Axilla (Hippocrates Method) This method is mentioned only to condemn it. The amount of traction and leverage that are used in its execution make it a formidable procedure. The number of catastrophes following its employment surpasses those of all the other methods combined. Nerve injuries are particularly likely to occur. Moreover, the procedure never is indicated either as a routine method or in isolated cases, because simple traction on the adducted humerus or the Kocher manipulation are capable of reducing all uncomplicated dislocations.

The patient lies on his back for a right-sided dislocation the operator places the bared right foot into the axilla close to the ribs. He grasps the patient's wrist in both

FIG 215 Hippocrates maneuver to reduce dislocations by traction and leverage with foot in axilla.



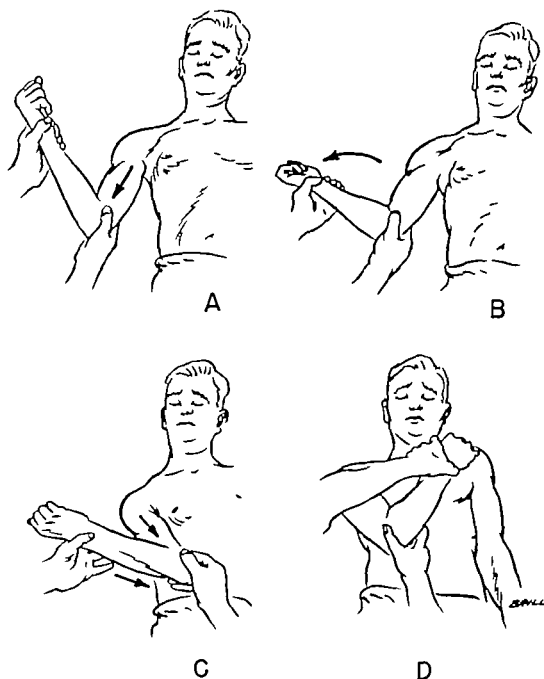


FIG. 214 Kocher's maneuvers to reduce dislocations of the scapulohumeral joint. (A) Preliminary stretching in the line of the long axis of the shaft of humerus in slight abduction. (B) The arm is externally rotated (C) the elbow is moved forward to a point near the midline of the trunk. (D) Finally the arm is internally rotated so that hand falls over the opposite shoulder

nerve are frequent catastrophes resulting from the injudicious use of this method

One is justified to risk the hazards of the Kocher method only when traction on the adducted humerus method has failed or in late dislocations when soft tissue organization and contracture demand the use of more forceful means to dislodge the humeral head and to replace it in the glenoid fossa

Before an extremity is put through the various Kocher steps there should be a preliminary stretching maneuver to bring the upward displaced head to the inferior margin of the glenoid cavity. The patient is placed on his back with the elbow flexed for a right-sided dislocation the operator grasps the lower end of the humerus above the epicondyles with his right hand and the

referable to the shoulder joint have not been emphasized sufficiently

The following clinical facts should govern the management of acute dislocations. Although this lesion is encountered more frequently in muscular individuals under 30, 'frozen shoulders' are rarely observed before the age of 30 and usually occur after middle life. Recurrences are common complications before 30 years, but they are rare after 45 years of age. Recurrent dislocation is, in a true sense, self limiting in nature. The author has three cases under observation aged 52, 57 and 61 years respectively. All have had frequent recurrences during youth but without any form of treatment none has had a dislocation after his forty fifth year.

It is obvious that some process is at work which over the course of many years, eventually stabilizes the glenohumeral joint. In the investigation on lesions of the shoulder joint it has been noted that after the third decade there is macroscopic and microscopic evidence that in the capsular tissues and the musculotendinous cuff progressive degenerative changes characterized by fibrosis thickening and loss of elasticity occur normally. This observation is sustained further by a study of 800 cases in various age groups which disclosed a gradual decrease in the range of external rotation in each subsequent decade (Fig 217).

One must conclude therefore that the afore mentioned process, which ultimately is responsible for restriction of external rotation also is the agent which restores stability. Nature's method to cure the malady is by restricting external rotation. The surgeon unknowingly accomplishes the same objective by numerous ingenious operative procedures.

Too much emphasis has been placed on trauma sustained by the labrum glenoidale, the humeral head and the fibrous capsule in relation to the pathogenesis of recurrent lesions. On the other hand there has not been enough emphasis on the trauma inflicted upon the muscular apparatus, par-

ticularly the rotator muscles, of the glenohumeral joint. It was noted in the pathology of acute dislocations that the most constant lesion was stretching and at times tearing of the short rotator muscles. It is the writer's opinion that such trauma initiates a neuromuscular state characterized by loss of tonicity and efficiency similar to that associated with stretch paralysis of muscles. When such a state persists the stabilizing effect of the rotator cuff is inadequate, and if the muscles are caught off guard or submitted to sudden severe strain while the arm is in some position of abduction and external rotation, recurrent dislocations ensue.

Frequency of recurrence is not dependent upon the degree of capsular relaxation, or the presence and the size of a defect in the humeral head or the severity of a "Bankartian lesion" (torn and detached labrum), but to the degree of inefficiency and loss of muscle tone of the short rotator muscles. This postulate is supported by observations made on shoulders obtained postmortem from individuals who prior to death had given no history of traumatic or recurrent dislocation. Lesions observed in the shoulders of these individuals were similar to those described by Bankart and others.

This fact leads one to conclude that the pathology noted by these observers is not the causative factor but only concomitant lesions of recurrent dislocation. They result from normal wear and tear and senescence, or they may be produced by trauma (see Chap 3). If the above concept is accepted it becomes mandatory that rest be an essential prerequisite for the restoration of normal muscular action and the efficiency of the short rotator group.

In accordance with the above reasoning one must modify the postreduction management to fit the individual case. As a rule in the treatment of young muscular individuals, one should be concerned primarily with the prevention of recurrences since stiffness due to prolonged fixation rarely occurs in the younger age periods. Immediate redislocation during the healing phase will not

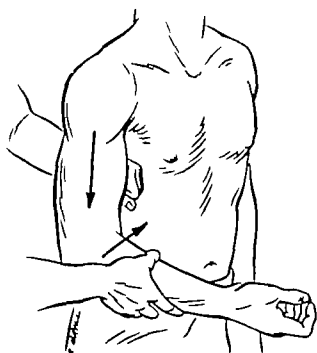
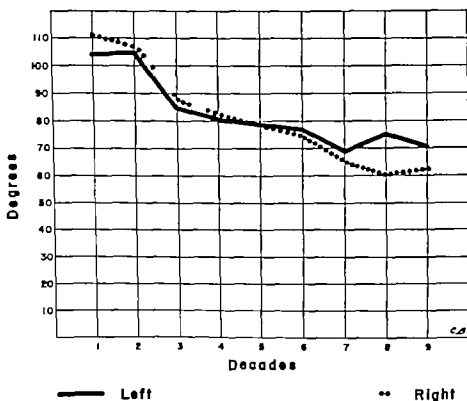


FIG 216 Nicolas maneuver, by making downward traction on the arm and by using the fist in the axilla as a fulcrum to lever the head over the glenoid brim

hands and makes slow steady traction in slight adduction. The foot is used as a fulcrum to lever the head over the glenoid brim. Slight outward rotation of the shaft may be necessary at times to complete the reduction (Fig. 215).

Post Reduction Management. There is still considerable disagreement as to the optimum position to maintain reduction whether or not the arm should be immobilized and when mobilization should begin. Some observers advocate fixation in abduction, others in a sling or some other comparable apparatus. Some advocate fixation of the extremity for one week, others for eight. It is obvious that this diversity of opinions is based on a lack of comprehension of the detailed pathology of acute dislocations and of the factors responsible for crippling sequelae and recurrences. Moreover, certain accepted clinical observations



Average range of external rotation at each decade

FIG 217 Graph showing gradual decrease in range of external rotation with advancing age. Study was made on 800 individuals, 100 in each decade from the first to the eighth. Only individuals who never suffered a dislocation or who never had severe injuries to the shoulder joints were selected for this investigation.

which the heads are near their anatomic positions and offer greater resistance to reduction, indicating less soft tissue disruption.

Gravity free stooping exercises of the extremity several times daily are started immediately (Fig 238). Care must be taken to move the limb within the painless arcs of motion—never to go beyond. Thus, healing tissues are not subjected to unnecessary deleterious strains. For the first 2 weeks abduction against gravity is restricted to 45°. Free usage of the arm within this safe range is encouraged. Within the following 2 weeks abduction is increased gradually to the horizontal position. All restricting apparatus is discarded after the fourth week, and gradual progressive return to complete function is allowed. Optimum functional results should be achieved in from 8 to 10 weeks.

COMPLICATIONS OF DISLOCATION OF THE SHOULDER

In general, the extent and the frequency of bone, capsular and nerve injuries associated with dislocation have not been fully appreciated. Detection of such lesions is a simple matter, provided that the examiner is aware of their possible existence and looks for them. Careful evaluation of the extremity by clinical methods and radiography both before and after reduction is essential in order to detect concomitant lesions. As previously stated their existence modifies the treatment of the dislocation.

The more common complications are

- 1 Fracture of humeral head (All types and combinations may occur)
- 2 Fracture of the glenoid rim
- 3 Rupture of the rotator cuff (The extent varies from incomplete tears to complete avulsion of the cuff)
- 4 Dislocation of the biceps tendon
- 5 Brachial plexus injury (The circumflex nerve is injured most frequently)
- 6 Vascular injuries.

The above lesions are considered in detail in Chapters 4, 8 and 9.

OPEN REDUCTION FOR INITIAL DISLOCATION

Conservative methods may fail to reduce what clinically appears to be an uncomplicated dislocation. Forceful maneuvers should not be employed when failure of reduction occurs. Less damage is inflicted if the joint is explored and inspected and if the lesion interfering with reduction is identified. If no fracture complicates the dislocation, the most common causes for failure of reduction are interposition of portion of the cuff or the inferior capsule between the glenoid fossa and the head of the humerus, or posterior displacement of the biceps tendon.

McLaughlin's superior approach (see Chap 11) is employed. It affords an excellent exposure of the entire subacromial region and the interior of the joint cavity with minimum risk to the axillary nerve. After the lesion responsible for interference is eliminated, reduction is effected by gentle traction on the adducted humeral shaft. If necessary, slight rotatory motions may be added. Longitudinal rents in the cuff are closed by interrupted side-to-side sutures. Transverse ruptures are repaired in the manner described in Chapter 4. Avulsion of the biceps tendon means disruption of the tendon-tendon sheath gliding mechanism and is likely to initiate a disabling tenosynovitis. In order to avoid such a complication, the author prefers to release the tendon at its insertion into the superior brim of the glenoid cavity and transplant it into the coracoid process (see Bicipital Tenosynovitis). The frequency of tenosynovitis of the biceps tendon following dislocation and fractures of the humeral head in the region of the intertubercular sulcus justifies this procedure.

If the head was forced out of a rent created by stripping of the inferior capsule from the neck of the humerus, no repair is necessary because spontaneous healing occurs readily in this region.

Postoperative management is that of

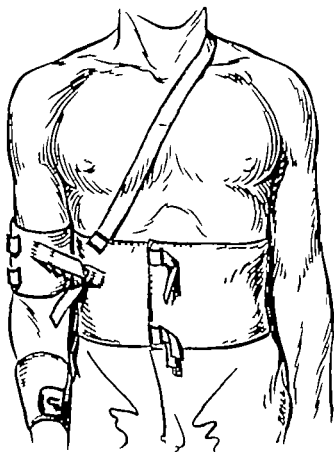


FIG 218 Nicola's apparatus used to immobilize the arm following acute dislocations in young individuals. It limits abduction and external rotation.

minutes. Later, as the range of motion and muscular power increases, wall-crawling and pulley exercises are added.

Free use of the arm is permissible within the limits of pain and fatigue. Physical therapy in the form of radiant heat and gentle massage of the entire shoulder girdle is a valuable adjunct, but the patient must be convinced that this form of therapy has no curative powers and that active motion is the key to rapid and normal restoration of shoulder function. Resumption of all activities and optimum function are attained within the next 4 weeks. Under the above regimen, sufficient time is allowed for complete return of muscle power, and tone and all capsular tissues are permitted to heal under minimum stress. Rehabilitation when started proceeds rapidly until complete function is attained.

The older age groups particularly the very aged present totally different problems. Recurrence of a dislocation rarely takes place; on the other hand stiffness must be avoided in the later decenniums. Clinical experience discloses that frozen shoulder is a frequent disabling complication following all traumatic lesions about the shoulder joint. The best treatment for this lesion is prevention, which can be accomplished by early mobilization within the tolerance of pain.

Immediately after reduction a sling or collar and-cuff is applied (Fig 237). This simple fixation suffices to maintain abduction and external rotation within safe limits, thereby preventing redislocation. Although early mobilization and rapid restoration of function are the aims of the surgeon, the rapidity with which they are attained must be governed by his evaluation of the case. Initial dislocations in which the heads are displaced at a great distance from the glenoid fossa and are reduced with relative ease indicate extensive soft tissue and capsular damage. Protection of these shoulders should be longer and restoration to full activity should be slower than those in

occur unless the extremity is hyperextended or abducted and rotated externally against gravity. Late recurrences occur with failure of the rotator muscles to return to normalcy. The best results in this group are obtained by immobilizing the extremity to the side by apparatus as described by Nicola (Fig 218).

It restricts elevation, abduction and all rotatory motions, yet allows a few degrees of forward and backward motion sufficient to promote absorption of debris within the traumatized joint. Motion at the elbow, the wrist and the hand should be encouraged. Fixation is maintained for 8 weeks, then a program of graduated motions is prescribed within the limits of pain and tolerance of the patient. At the beginning motion should be performed in a stooped position every hour during the day for from 5 to 10



FIG 219 (*Left*) Characteristic features of the normal glenohumeral articulation with the head of the humerus rotated externally. Note the prominent lateral border of the head formed by the greater tuberosity, the lesser tuberosity is medial and parallel to it, while the intertubercular sulcus is visualized between the two. The glenoid fossa casts a crescent shadow over the medial articular surface of the head. The rounded articular surface of the head covers the inferior region of the glenoid fossa. (*Right*) Characteristic features with the humerus rotated internally. The greater tuberosity is barely distinguishable, while the lesser appears to be in the glenoid cavity. The inferior portion of the glenoid fossa is covered by the tuberosities. Note that the rounded articular surface of the head is directed upward and outward.

In internal rotation the tuberosities are seen on the medial side of the humeral shaft. In fact, the lesser tuberosity appears to be in the glenoid cavity, and the greater tuberosity is barely distinguishable, the rounded articular surface of the head being now directed upward and outward. At the same time the glenoid fossa casts a crescent shadow over the tuberosities (Fig 219, *right*).

In anteroposterior views of a posterior dislocation the afore mentioned normal features are greatly distorted. The humeral shaft lies in full internal rotation. However, the lateral margin of the head is formed by the lesser tuberosity while the greater tuberosity medial to it is barely visible. The inferior region of the glenoid fossa is now

exposed and no longer covered by the spherical articular surface, as in normal external rotation and by the tuberosities in normal internal rotation (Fig 220).

Lateral views obtained by placing the cassette on the abducted shoulder and directing the rays into the axilla are more reliable than those directing the rays through the thorax with the cassette on the lateral aspect of the dislocated shoulder.

Under general anesthesia reduction is effected readily by gentle steady traction on the arm with the elbow flexed in the long axis of the humerus. When the head reaches the brim of the glenoid fossa, gentle adduction and internal rotation of the arm complete the reduction. Pressure on the head from behind may facilitate the reduction.

simple, uncomplicated dislocation reduced by conservative methods but should be tempered by the surgeon's knowledge of the extent and the severity of the soft tissue injuries observed at operation

LUXATIO ERECTA

Reduction of this lesion is effected readily by traction on the arm directed upward and slightly outward. Countertraction, if desired, may be provided by a sheet across the supraclavicular region pulling downward. Reduction is indicated by an audible snap, as the head glides over the rim and engages the glenoid fossa. The arm is then brought gently to the side. Postreduction treatment is similar to that of subcoracoid dislocation.

ACUTE TRAUMATIC POSTERIOR DISLOCATION

This type of dislocation is extremely rare and frequently it is unrecognized. Depending upon the location of the humeral head it is classified either as subacromial or subspinous. Usually its mechanism of production is one of forceful dorsal flexion with the arm rotated internally. Cases have been reported in which the head was displaced backward by a direct force on the anterior aspect of the shoulder joint. Epileptic convulsions is a frequent cause of posterior dislocation. The author has seen one case following electric shock therapy.

Most writers note that the head is forced through a rent in the posterior capsule. In the literature however this conception lacks confirmation of acute cases explored with the head in a posterior position. Codman noted the findings of one case of an old unreduced subspinous dislocation in which he recorded that none of the short rotators was ruptured. Robertson and Stark observed after operating on three cases of recurrent posterior dislocation that in every case the capsule was redundant and lax.

Like Rowe and Yee, Hlindenach failed to find a detached labrum glenoidale. Rowe and Yee noted in two cases that the capsule had been stripped from the neck of the

scapula. All the above recurrent lesions followed initial traumatic dislocations. It is logical to assume from the observations made in anterior dislocations that the humeral head in posterior lesions may be forced through a rent in the musculotendinous cuff, or they may strip the labrum, the capsule and the rotator muscles off the posterior surface of the neck of the scapula and lie beneath them in an intracapsular position. Regardless of whether the head is intracapsular or extracapsular, it is obvious that the soft tissues are stretched or torn severely. Damage of varying severity may also be inflicted upon the head and the glenoid rim.

Although the diagnosis is made readily, the great amount of swelling around the shoulder joint may confuse the clinical picture. The anterior region of the shoulder is flattened while the acromion and the coracoid processes are prominent. A globular mass is palpable posteriorly which rotates with the shaft. The arm is held in adduction occasionally depending on the position of the head as in a subspinous position it may be slightly abducted and forward. All motions are painful.

Roentgenographic examination in the anteroposterior view may fail to demonstrate the lesion but lateral views depict clearly the relation of the head to the glenoid fossa. Failure to recognize the lesion in anteroposterior views can be attributed chiefly to unfamiliarity with the appearance of the glenohumeral joint in various degrees of rotation.

In external rotation the greater tuberosity forms the prominent lateral border of the humeral head the lesser tuberosity being medial and parallel with it. The intertubercular sulcus and the bicipital groove are visualized clearly between the two. The medial border of the head is formed by its smooth hemispherical articular surface over which the superimposed glenoid fossa casts a crescent shadow. The rounded articular surface of the head covers the inferior region of the glenoid fossa (Fig 219 left).

better results than the open procedures. Cases which are irreducible by the ordinary closed methods properly executed usually have existed for a long time, and profound secondary changes of the affected tissues have taken place.

PATHOLOGY

In addition to the tissue damage recorded in the pathology of anterior dislocation, advanced secondary changes characterized by tissue infiltration and organization are found in the late lesions. Depending upon the duration of the lesion, there are varying degrees of fibrosis, contracture and shortening of all involved tissues particularly the musculotendinous cuff and the fibrous capsule with scar tissue formation firmly fixing the humeral head in its unnatural site to the neighboring structures. The long adductors and the internal rotators (*pectoralis major*, *teres major* and *latissimus dorsi* muscles) also became fibrotic and shortened. Tough, inelastic scar tissue fills the synovial cavity, obliterating it completely. In very late cases the articulating cartilage of the glenoid fossa and the humeral head undergoes fibrillation, thinning and, finally, erosion and complete disintegration. Loose bony fragments, if present, become embedded in fibrous tissue. Profound demineralization and bone atrophy may be discernible in the glenoid, the humeral head and the shaft. In very old cases bony proliferation appears on the glenoid rim and at the point of contact of the head against the scapula. The biceps tendon not infrequently is traumatized severely. It may be severed completely its distal end having reattached itself to the humeral shaft below the lesser tuberosity. In two instances it was displaced posteriorly, thereby preventing reduction.

It is apparent that success of ordinary closed methods depends entirely on the extent of the initial trauma and the degree of superimposed secondary changes.

COURSE OF UNTREATED CASES

Although some observers have recorded

cases in which pain was insignificant and functional capacity of the shoulder fair, usually this lesion is associated with considerable pain and pronounced disability. In most instances, pain is not lessened, nor is function improved with time. In some late cases (if pain is not severe and the functional adaptation is fair) no interference other than physical therapy is indicated to improve the muscular tone and the range of motion of the extremity. This is particularly true in elderly individuals. However, if pain is pronounced and the functional adaptation is poor, attempts at reduction are justifiable, especially in younger individuals in whom no other contraindications exist.

CHOICE OF TREATMENT

To accept the dislocation and to strive for as much function as possible by physical therapy is justifiable in elderly individuals in whom pain is not a prominent factor, and also in patients for whom manipulative attempts at reduction have failed and operative intervention is contraindicated.

Closed manipulative methods should be attempted in dislocations under 12 weeks' duration. Not infrequently physical examination discloses marked contracture and shortening of the adductors and the internal rotators. In such instances, preliminary skeletal traction through the olecranon in the line of the axis of the humerus may facilitate the reduction.

Reduction by open methods is indicated in all dislocations over three months duration and when reduction has failed by the closed procedures.

Arthrodesis of the glenohumeral joint though a radical procedure may be necessary in the presence of advanced disintegration of the articulating surfaces of the glenoid and the humeral head. It is preferable in younger individuals when because of severe degenerative changes in the affected tissues it is obvious that simple replacement of the humeral head will not result in a very serviceable shoulder joint. Consideration of the earning capacity of



FIG 220 Posterior dislocation Note the loss of outline of the greater tuberosity The lower portion of glenoid cavity is exposed and no longer covered by the spherical articular surface of the head in normal external rotation and the tuberosities in internal rotation.

As a rule the latter maneuvers are not necessary Postreduction treatment is the same as for anterior dislocations

Most posterior dislocations are followed by no disturbing sequelae If reduced properly and early but recurrent dislocations occasionally ensue At times the reduction after the initial trauma is difficult to maintain. J C Wilson and F M McKeever have devised an ingenious operation to stabilize the glenohumeral joint after reduction (Fig 221) The acromion and the head of the humerus are fixed by two wires crossing one another The wires are drilled through the top of the acromion and through the head They are left protruding from the skin over the acromion and are removed in three weeks Wilson and McKeever advocate this

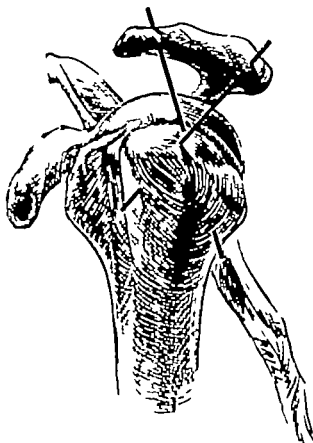


FIG 221 Method to maintain position following reduction of posterior dislocations which tend to recur (Wilson and McKeever)

type of fixation also for old posterior dislocations after reduction is attained by surgical intervention.

OLD DISLOCATIONS

Unreduced dislocations of the shoulder joint tax the skill of the most experienced and skillful surgeons. Adequate management is influenced by many factors such as the age of the patient, the severity of the symptoms the degree of disability and the duration of the lesion Treatment based on faulty evaluation of these factors is doomed to failure and may be responsible for irreversible damage to the brachial plexus, the axillary artery and vein the musculotendinous cuff and the bony components of the glenohumeral joint. Prognosis should be guarded in all old dislocations. In general, it is better in the early than in the late cases and successful closed methods gave

head ensue. Such a complication will cause a stiff and painful shoulder in spite of good reduction. Posterior displacement of the biceps tendon may be a formidable obstacle to reduction. In such instances, the tendon is divided, and its proximal end is passed through the humeral head and reattached to the distal end, as in a Nicola procedure.

Reduction having been accomplished, attention is directed toward repairing the capsular defect. The divided tendons (subscapularis and pectoralis major) are resutured, and, if an opening in the capsule remains, some type of repair must be attempted to prevent subluxation or dislocation.

Maintenance of the reduction may prove to be difficult. A Nicola operation may ensure stability of the joint. Cubbins has utilized the coracohumeral ligament for this purpose. The ligament is cut close to its coracoid attachment and freed down to the humeral insertion. The proximal free end is then passed through a drill hole in the acromion and anchored to the periosteum on its superior surface. The transposed structure then functions as a suspensory ligament of the humeral head.

Neviaser maintains reduction with a vitallium transfixion screw passing through the head into the glenoid cavity. The screw is removed after 3 or 4 weeks (Fig. 222).

The method of stabilization for recurrent posterior dislocations by Wilson and McKeever appeals to the writer as a method worthy of trial in maintaining reduction after it is effected in old dislocations. These workers transfix the humeral head and the acromion with cruciate wires, the ends of which are left protruding from the skin over the superior surface of the acromion. They are removed after 3 weeks.

In cases in which no internal fixation is used to stabilize the joint postoperative immobilization with plaster or splint is necessary. The most stable position of the arm is from 60° to 80° elevation midway between the coronal and the sagittal planes. Fixation is continued from 4 to 6 weeks and

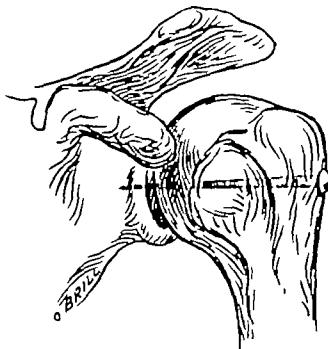


FIG. 222 Neviaser's method of maintaining normal position after reduction of old dislocations of the scapulohumeral joint.

followed by intense physical therapy. Graded active exercises (first gravity free and later against gravity) are the most important features of this phase of the treatment.

The result may be considered satisfactory if the patient is free of pain and can abduct the glenohumeral joint 60° with good muscle power. Weak abduction in some cases may be the result of axillary nerve injury at the time of the initial trauma or it may result from pressure of the dislocated humeral head on the nerve, or injury at the time of operation.

RECURRENT DISLOCATION OF THE GLENOHUMERAL JOINT

The lamentably high percentage of failures of popular stabilizing procedures for recurrent dislocation has been in recent years a stimulus to investigate further the pathogenesis and the management of this lesion. Hitherto the profession at large was inclined to accept as final, observations made without further investigation of the problem. Also there has been a tendency to

patients in the younger age group should influence the surgeon's ultimate choice. A painless arthrodesed glenohumeral joint is by far more serviceable than a painful joint with from 30° to 60° of abduction.

Resection of the humeral head occasionally must be performed. If extensive stripping of the soft tissues is necessary to mobilize the head, aseptic necrosis of the head of the humerus invariably ensues, resulting in a stiff and painful shoulder. Resection of the head in such instances is justifiable. This may or may not be followed by arthrodesis depending upon the age and the activity of the patient. A flail shoulder joint, while painless, is not very serviceable but it may suffice for aged individuals.

CLOSED METHODS

All manipulative maneuvers used in old dislocations even in the hands of the skillful, are dangerous and may produce extensive damage to the brachial plexus and the axillary artery, or they may fracture the humerus. The operator must be cognizant of these potential hazards and be ready to accept such complications. In view of the existing pathology of necessity all maneuvers must employ considerable force the extent of which must be tempered by the skill and the experience of the surgeon.

Complete relaxation is essential and is best attained by general anesthesia. Before definitive maneuvers are employed to effect reduction the humeral head is mobilized by steady traction on the arm with the elbow flexed. The shaft is then rotated inwardly and outwardly slowly and forcefully thereby freeing the head in its bed from all adhesions. Next, while traction is maintained the arm is abducted slowly and rotated externally. Such maneuvers overcome contracture and shortening of the long and the short adductors and the internal rotators particularly the pectoralis major and the subscapularis muscles.

Following the foregoing preliminary maneuvers replacement of the humeral head is attempted by the Kocher method. Failure

to attain a reduction justifies another trial by the same or a different operator, but repeated attempts at reduction are condemned. Every attempt is accompanied by extensive soft tissue damage and increases the risk of severe complications.

Failure to effect a reduction leaves the operator one of two choices, either to accept the disability and try to regain the maximum function possible by physical measures or, at a later date, to resort to open methods.

OPEN METHODS

Bennet emphasizes several pertinent points in the technic of open procedures which facilitate repositioning of the humeral head: adequate exposure of the area, cleaning the glenoid cavity of all scar tissue, and severance of the subscapularis tendon.

The author has found that the best exposure is afforded by the incision of Cubbins, Callahan and Scuderi (Fig. 337). By careful dissection, the joint capsule and the humeral head in their abnormal position are identified. Upon external rotation of the humerus, the subscapularis muscle is visualized and cut about one half an inch from its insertion into the lesser tuberosity. Retraction of the muscle medially provides adequate exposure of the glenoid fossa which is now cleaned of all accessible scar tissue. Care must be taken not to injure the axillary nerve when the subscapularis tendon is divided. This mishap can occur readily and occasionally does.

In most instances, severance of the subscapularis muscle permits sufficient mobilization of the head to effect a reduction. Occasionally it may be necessary to divide part or all of the insertion of the pectoralis major. As a rule use of the Kocher method permits the head to be restored with comparative ease to its normal position in the glenoid fossa. If necessary a bone skid may be used to lever the head into position. Extensive stripping of the capsular attachments from the humeral head and neck must be avoided lest aseptic necrosis of the

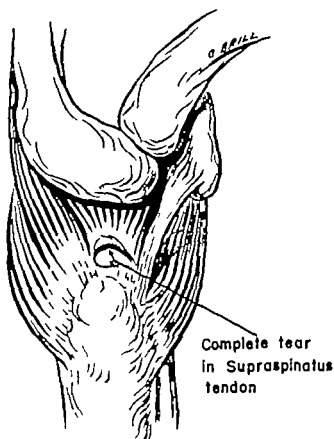


FIG 224 Small complete supraspinatus tear found in 2 of 56 cases of recurrent dislocation explored.

posterior views with the humerus in 50° to 80° of internal rotation. It is apparent that difficulty in demonstrating the lesion is responsible for the wide discrepancies recorded in the literature. Adams reports an incidence of 82 per cent.

Hill and Sacks describe the defect as a flattening or depression of the posterolateral region of the articulating surface of the humeral head. Roentgenographically, it is visualized as a depression in the upper and outer quadrant of the spherical head of the humerus (Fig. 223). Frequently a line of increased density is observed extending distally from the top of the humerus along the mesial margin of the defect and parallel with the humeral shaft.

Those who are convinced that the above defects in the humeral head are capable of producing dislocation also believe that the frequency of the lesion depends upon the size of the lesion. As the defect increases in

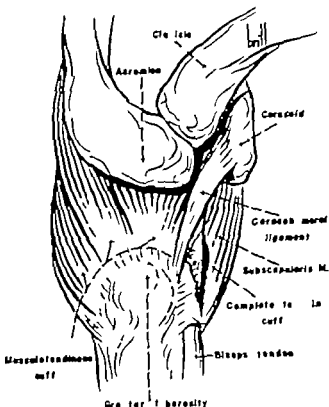


FIG 225 Small complete tear found in 3 of 56 cases of recurrent dislocations explored.

size the tendency to dislocation becomes greater.

Both lesions, detachment of the labrum glenoidale and defects in the humeral head may exist in the same shoulder or independently. In a comprehensive study, Adams recorded detachment of the labrum from the glenoid rim (Bankartian lesion) in 87 per cent of the 79 shoulders explored. 13 per cent disclosed normal attachment of the labrum. Of 68 cases studied, 56 (82 per cent) disclosed bone defects in the humeral head. He noted that when a labral defect was not discernible, the head of the humerus disclosed a bone defect in its posterolateral region.

Pathologic Observations. The following pathologic observations were made in 56 recurrent dislocating shoulders of 56 individuals. There were 55 males and 1 female, their ages ranging from 19 to 33 years. The woman was 33 years old. In all instances the initial lesion resulted from some form of violent injury. However the author has seen 4 cases not included in this study in



FIG. 223 Defect in the upper and outer aspect of the head of the humerus of a patient with recurrent dislocations.

designate some local abnormality in the glenohumeral joint as the causative factor of the lesion.

When the pathology of recurrent dislocation is considered most workers fail to evaluate the glenohumeral joint in its entirety restricting themselves to some local irregularity of the labrum glenoidale the humeral head or the fibrous capsule. We are taught that the stability of the glenohumeral joint is directly dependent upon the surrounding intricate muscular apparatus that motivates the joint. Yet when a state of instability exists local defects in other components of the joint are credited as causative agents.

PATHOLOGY

Numerous theories have been offered as the underlying causes for recurrent dislocation. Bankart stoutly defends his belief that the pathology in all instances is either a detachment of the labrum from the anterior

glenoid rim or a tearing away of the capsule from the labrum. He goes so far as to express the view that the recurrent lesion is an entirely different entity from the ordinary acute traumatic dislocation. The former, according to his view, is invariably an anterior dislocation which tends to recur because the fibrocartilaginous labrum fails to reattach itself to the glenoid margin. The latter is an inferior dislocation, the head being forced through a rent in the fibrous capsule which heals readily to bone, thereby preventing recurrences. These features of the morbid anatomy of recurrent dislocation were recorded by Broca and Hartmann as far back as 1890. These observers also noted a defect on the posterior aspect of the humeral head which they believed facilitated intracapsular subluxation of the head of the humerus. Many other workers described the above labral and capsular defects and concentrated their efforts in repairing them in order to effect a cure of the lesion.

Since the first report on defects of the humeral head in relation to recurrent dislocation by Broca and Hartmann other investigators have confirmed this abnormality and stated that it plays a major role in the production of recurrences (Fowler, Schultze, Pilz, Holland, Sacks and Adams). According to Adams this causative factor has not been generally accepted because of the difficulties in visualizing the defect by routine anteroposterior roentgenograms, and exposing the lesion at operation by the anterior incision commonly employed.

Hill and Sacks are of the opinion that the defect is a compression fracture of the spongy bone in the posterolateral region of the head of the humerus sustained by forceful impact of this area against the glenoid rim at the time of the initial dislocation. They contend that the lesion is only demonstrable roentgenographically when anteroposterior views are taken with the arm in internal rotation or in tangential projections. Adams demonstrates clearly that defects are most often demonstrable in antero-

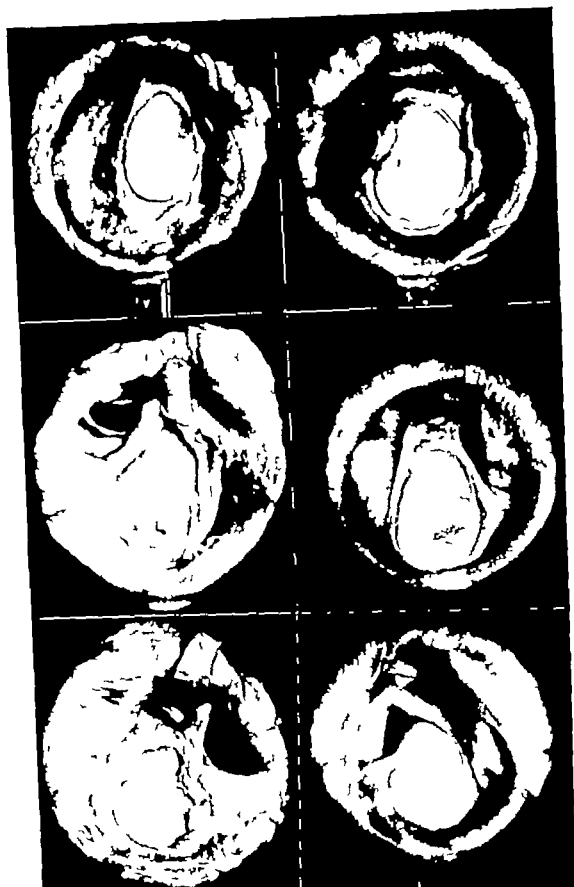


FIG. 227 Types of subscapularis recesses found in 88.6 per cent of the shoulders studied in the investigation on variational anatomy and degenerative lesions of the shoulder joint. Note that in the anterior aspect of the joint the fibrous capsule is not continuous with the labrum but is projected medially to the subcoracoid region and then reflected onto the neck of the scapula to blend with the labrum.

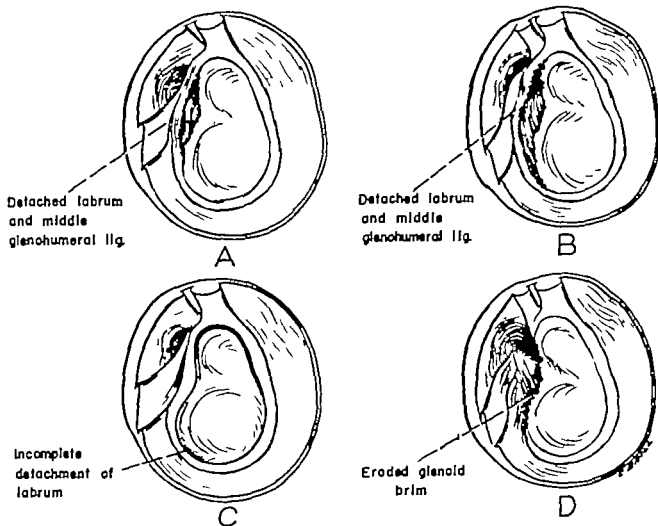


FIG. 226 Varying degrees of labral detachment found in series of recurrent dislocations investigated surgically. Eighty five per cent exhibited lesions (A, B and D). In 15 per cent, the labrum and capsule were intact (C).

which the initial dislocation resulted from trivial injuries. Two others also excluded from this series recalled no trauma whatsoever. The lesion was bilateral in 2 of the 6 cases.

Musculotendinous Cuff. An observation of considerable significance was the pronounced laxity of the cuff. It appeared as if the short rotators were unduly stretched and lacked normal tonicity. In 2 instances (3.5 per cent) small tears not over 1 cm. wide were demonstrable in the supraspinatus region of the cuff. The margins of the defects were smooth indicating that the lesions were not recent. Three shoulders (5.3 per cent) disclosed partial detachment of the inferior portion of the subscapularis

tendon at its insertion into the lesser tuberosity (Figs. 224 and 225).

Labrum Glenoidale and Fibrous Capsule. Some degree of detachment was discernible in 48 cases (85 per cent). The detachment was invariably from the anterior or antero-inferior portion of the glenoid rim. All degrees of detachment were noted varying from 1 or 2 cm. in length to complete detachment of the entire anterior half of the fibrocartilaginous ring. In most instances the capsule and the periosteum, together with the labrum were stripped for varying distances from the anterior surface of the neck of the scapula. This last feature was more pronounced in cases with extensive labral detachments. Such a defect com-

Bone Changes. Varying degrees of erosion and eburnation were found on the anterior lip of the glenoid fossa in cases in which there were labral detachments. In one instance, a large irregular defect was demonstrable, leading to the conclusion that a fragment of bone had been sheared away from this area (Fig 228). Some osteophytes were observed on the anterior surface of the neck in three cases with extensive labral detachment.

Thirty seven of the 56 cases were studied roentgenographically with the humerus in varying degrees of internal rotation. Twenty three (62 per cent) disclosed evidence of a defect in the posterolateral region of the humeral head. No lesion was demonstrable in 14 cases (38 per cent).

A comparative analysis of the pathology observed in recurrent dislocation and in initial dislocation discloses very little dissimilarity. In the recurrent lesion there is added evidence of a repair, fibrosis and secondary changes in soft and bone tissues. It was of great significance to note the paucity of complete cuff tears in recurrent lesions as compared with the frequency of these lesions in primary dislocations, indicating that a fresh dislocation is followed by very active reparative processes.

Discussion. Many observers are of the opinion that labral and capsular detachments from the anterior glenoid rim and the capsular neck are essential causative factors in recurrent dislocations. A smaller group contends that the defect in the humeral head plays the major role in producing this lesion. Yet others maintain that both lesions may be responsible for the tendency to recurrences. The author is unable to accept these concepts as the true causative factors in recurring dislocation because other observations fail to support such premises.

In Chapter 3 it was recorded that some degree of labral detachment was a frequent observation after the third decade, the extent and the severity of this abnormality increasing with each subsequent decade.

After the sixth decade it was demonstrable in approximately 100 per cent of the specimens.

The severity and the frequency of the lesion was the same, both in a series of cadaver specimens and in a series of post mortem specimens in which a negative shoulder history had been obtained prior to death. From this observation alone it is reasonable to assume that detachment of the labrum is associated with advancing age. Moreover, its intimate relationship to the glenohumeral ligaments and the biceps tendon render it vulnerable to stresses, chiefly distracting forces, which play a part in its detachment from the glenoid rim.

Clinical experience teaches that recurrent dislocations are common in the earlier decades but rare after the fourth. Yet, the afore-mentioned investigation reveals that in the decades in which labral detachment are most frequent and severe recurrent dislocations are rarely encountered. In the light of these observations one is forced to conclude that some agent other than labral and capsular lesions as described by Bankart and many others, plays a greater role in the tendency to recurrent dislocation.

As previously noted, recurrent dislocation is a self limiting disease. Many individuals had in early life numerous recurrences which without treatment, steadily decreased as the individuals approached middle life. Finally no more occurred. This is readily understandable when one recalls that the musculotendinous cuff exhibits manifestations of a degenerative process characterized by fibrosis of all soft tissue particularly the subsynovial layers and fibrous capsule shortening and loss of elasticity as early as the third decade. Such features increase in severity from decade to decade. Figure 217 represents graphically the gradual decrease from early to later decades in the range of external rotation. It is obvious that the same process which is responsible for loss of external rotation also cures recurrent lesions and restores stability of the glenohumeral joint.

prises the classical Bankartian lesion" Eight cases (15 per cent) disclosed the labrum to be firmly anchored to the glenoid margin by its capsular border however its glenoid border was free like a meniscus

It becomes obvious that it is impossible for the head to pierce the capsule in the anterior portion of the joint unless it is forced through a rent in the anterior portion of the cuff or through a rent in the inferior

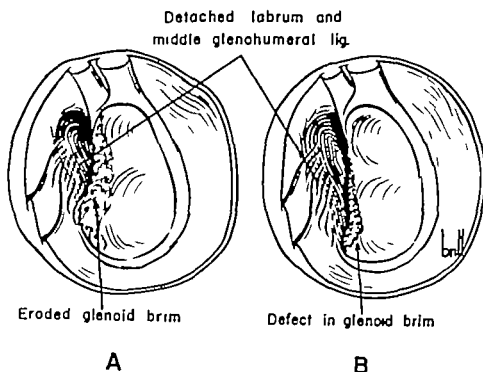


FIG 228 (A) Bony alterations observed along the anterior lip of the glenoid cavity (B) A large defect in the anterior and inferior portion of the glenoid cavity suggests the possibility that a piece of bone was sheared away from this region

Many of the detached labra disclosed advanced fraying shredding and thinning (Fig 226)

At this point it should be recalled that the fibrous capsule in the anterior portion of the glenohumeral joint is not as so many believe continuous with the labrum but is projected mesially as far as the subcoracoid region and then reflected onto the neck of the scapula until it reaches the periphery of the labrum This outpouching of the capsule forms the subscapularis recesses one or two depending upon the anatomy of the variable middle glenohumeral ligament. Such an arrangement was observed in 88.6 per cent of the specimens studied, only 11.4 per cent revealed the capsule continuous with the labrum and in these there were no subscapularis recesses (Fig 227)

capsule below the lower border of the subscapularis muscle The head therefore, in recurrent dislocations lies in the subscapularis recess or recesses which are stretched to accommodate it The middle and the inferior glenohumeral ligaments may be the only feeble barriers to dislocation.

Glenohumeral Ligaments and Subscapularis Recesses. In many instances the middle ligament could not be identified. In a few small shreds of tissue indicated the remains of the ligament. In most shoulders the inferior ligament was discernible, but it was greatly attenuated and stretched All shoulders revealed marked stretching of the subscapularis recesses and one could pass a probe mesially on the anterior surface of the neck of the scapula as far back as the coracoid process

are not infrequent postoperative complications.

By this method, the intracapsular portion of the biceps tendon is converted into a suspensory ligament for the head of the humerus. At this point it should be recalled that the biceps tendon is continuous with the posterior and the anterior portions of the labrum glenoidale. Also as the result of normal wear and tear and the distracting influence of the biceps tendon on the labrum, there is a gradual separation of this structure from the glenoid rim. This is manifest as early as the second decade and increases in severity in each subsequent decennium.

Therefore it becomes readily understandable that the added pull upon the labrum subsequent to suspension of the humeral head on the biceps tendon enhances labral detachment from the glenoid cavity thereby defeating the very purpose for which the operation is performed. In the author's opinion labral detachment is the most common cause for failure of the Nicola procedure. This feature was clearly demonstrable in 6 instances of redislocation following a Nicola operation. Another technical error that may be responsible for failure is placing the drill hole too close to the periphery of the articular surface of the humeral head. Disintegration or rupture of the new suspensory ligament may be responsible for some failures.

Henderson's tenosuspension operation, rather popular at first is now used less frequently. A high proportion of failures follows this procedure. Pain (1948) recorded that recurrences took place in 9 of 13 cases in which a fascial or tendon sling operation was done. Redislocation occurred in every case in which a fascial sling was employed and in 4 of 7 cases in which the peroneus longus tendon was used. On the other hand, Henderson (1943) reported good results in 94 per cent of the cases in his series.

BONY BLOCK OPERATION. The mechanical objective of these procedures is to produce in front of the humeral head a bony block which prevents the head from being levered

out of the glenoid fossa. Speed used a tibial graft for this purpose, while Eyre Brook utilizes an iliac graft. Oudard, in addition to shortening the subscapularis tendon inserted between the tip and the base of the coracoid process a bone graft which extended in front of the glenohumeral joint or turned down in front of the subscapularis half of the coracoid process. All these procedures are formidable and technically difficult.

PLASTIC PROCEDURES ON THE CAPSULE AND THE TENDONS. All plastic procedures aim to build a soft tissue barrier in front of the humeral head, thereby preventing its displacement out of the glenoid cavity. Gallie and LeMesurier construct a new ligament of fascia, which later passes through drill holes in the scapula and the head of the humerus. They report only 7 redislocations in 175 cases.

Bankart conceived his operation on the premise that labral detachment is the "essential lesion" responsible for redislocation. He attempts to restore the normal anatomy of this region by suturing the labrum, or, if the labrum is attenuated he sutures the capsule to the scarified glenoid margin. Statistical studies of end result studies from numerous sources reveal a high percentage of cures. Bankart himself never has had a failure and attributes reported recurrences to technical errors.

The operation is difficult and should be performed only by surgeons who are familiar with the topographic anatomy of this region. External rotation is definitely restricted by this method. Adams notes in 18 cases the average range of external rotation to be 51°. The author is in agreement with many other observers that the success of the procedure depends upon the resulting restriction of external rotation and not on the reattachment of the capsule or labrum to the rim of the glenoid fossa.

The Putti Platt operation which was performed first by Valtancoli in 1925, is simpler than the Bankart operation and produces equally good results. Here again

PATHOGENESIS OF RECURRENT DISLOCATION OF THE SHOULDER

The writer is of the opinion that capsular, labral lesions and humeral head defects are not the prime causative agents of recurrent dislocation. They are changes associated with aging but they may be produced or aggravated by trauma. Neuromuscular imbalance chiefly of the short rotator muscles, is the most important single causative factor. Such a neuromuscular state follows severe stretching of, and direct injury to, the involved muscles (particularly the subscapularis muscle) at the time of injury. Pronounced capsular stretching and enlargement of the bursal recesses to accommodate the head are secondary adaptive changes. Repeated dislocations increase the neuromuscular imbalance hence increasing the tendency toward recurrence until nature overcomes the laxity of all tissues by a progressive process of fibrosis, which limits external rotation and stabilizes the glenohumeral joint.

If the afore mentioned concept of the pathogenesis of recurrent dislocations is accepted it becomes apparent that such a complication may follow any initial mechanism of anterior dislocation provided that the short rotators particularly the subscapularis muscle are severely stretched and traumatized and that the primary dislocation has not been treated adequately. The writer is in total disagreement with Bankart's postulate that recurrent dislocation is a different lesion than ordinary acute traumatic dislocation and that it can occur only in the presence of capsular or labral detachment. An essential or Bankartian lesion does not exist.

Capsular and labral lesions as well as defects in the humeral head have been given an undeserved place of importance in the pathology of recurrent dislocation. It has been revealed clearly that they are normal degenerative changes associated with wear and tear and senescence—although it must be admitted that trauma plays a part in their formation.

TREATMENT

Prevention. Adequate and prolonged immobilization of the arm in internal rotation in young individuals is an essential phase of the treatment of initial dislocations (see Treatment of Acute Anterior Dislocations).

Operative Procedures. Numerous surgical techniques have been devised to stabilize the glenohumeral joint. Some of these are simple operative procedures others are difficult. Excellent results have been claimed for all the different methods of approach yet, critical analysis of the end results discloses an alarmingly high percentage of failures. Evaluation of the mechanical principles upon which each procedure is based allows the following conclusions.

SUSPENSION OPERATIONS. The mechanical objective of such procedures is to create a suspensory ligament for the humeral head from fascia or a tendon which will confine the movements of the head within the glenoid cavity thereby preventing subluxation or dislocation.

Nicola's operation has been greatly popularized because of its simplicity. Reliable end result studies have revealed that this procedure has not come up to the expectations of its supporters. Hobart (1938) noted 8 recurrences in a series of 132 Nicola procedures. Adams (1948) reported 21 failures in 59 Nicola operations (36 per cent). Many surgeons believe that this procedure is the one of choice in adults not engaged in strenuous occupations.

The writer feels however that this procedure never is indicated because it is mechanically and anatomically unsound. Moreover pain is a frequent distressing sequel to the Nicola operation. The pain undoubtedly results from direct injury to the articular cartilage of the humeral head or to the interposition between the articular surfaces of the ligament, which gives rise to an internal derangement of the shoulder not unlike that observed in the knee joint with a torn meniscus. Then too disturbing snapping and clicking sensations within the joint during certain movements of the arm,

its original insertion onto the humeral shaft (Fig 229)

Magnuson Operation (modified) An S-shaped skin incision is made on the anterior aspect of the shoulder, beginning at the inferior margin of the acromioclavicular joint. The interval between the deltoid and the pectoralis major is developed, taking care not to injure the cephalic vein, which is retracted medially with the pectoralis major muscle. By external rotation of the shaft of the humerus, the subscapularis tendon comes into view as it inserts into the lesser tuberosity. A blunt dissector is passed under the subscapularis tendon in order to determine more clearly its upper and lower borders. An incision is made in the interval between the supraspinatus and the subscapularis muscles beginning proximal to the blending of the subscapularis tendon with the fibrous capsule and the incision is continued to the anterior lip of the bicipital sulcus.

A second incision the same length as the first is made along the lower border of the scapularis muscle. The tendon between the two incisions is then freed from the anterior lip of the bicipital groove by sharp dissection. Retraction medially of the tendon and the capsule affords a clear view of the humeral head, the anterior glenoid margin and the anterior portion of the synovial capsule with its glenohumeral ligaments and bursal recesses.

The greater tuberosity is visualized by internal rotation of the arm. By means of a suture through its substance, the subscapularis is then pulled across the bicipital groove to a point below the greater tuberosity and its site of reattachment is determined. The tendon should be anchored to the humeral shaft below the level of its original insertion under moderate but not severe tension. With a thin osteotome a slot one-quarter of an inch wide and as long as the width of the subscapularis tendon is made parallel with the posterior lip of the bicipital groove below the greater tuberosity. Three or 4 fine drill holes are then

made in the posterior lip of the newly formed slot, and the end of the tendon is buried in the bony trough with silk mattress sutures. The upper border of the subscapularis muscle is approximated to the supraspinatus muscle by side-to-side sutures, while its lower border is sutured to the capsular tissues under the head of the humerus.

Considerable restriction of external rotation is demonstrable at the completion of the procedure. The subscapularis muscle and the tendon fibers can also be seen to form a firm sling under the head on abduction of the arm in internal and external rotation. The procedure is completed by wound closure in layers with interrupted sutures.

POSTOPERATIVE TREATMENT For the first two weeks, the arm is fixed to the side in a plaster-of-Paris shoulder swathe (Fig 198). For the next 2 weeks the arm is kept in a sling. Motion is then begun, but not permitted above the horizontal plane for 2 more weeks. Forceful external rotation during this period is prohibited, but full resumption of all motions should be attained by the eighth week. As a rule, abduction is restricted a few degrees, and external rotation may be restricted as much as 50 per cent. However, this produces no functional disability and is assurance against redislocation. The amount of external rotation lost is a small price to pay for the cure of such a disabling malady by so simple a surgical procedure.

Bankart Procedure (Cave and Rowe) Exposure of the anterior region of the glenohumeral joint is obtained by the straight deltopectoral incision or by the S-shaped incision described by Hitchcock and Bechtol. Even better visualization of the entire cuff and inside of the glenohumeral joint is obtained by McLaughlin's transacromial incision. The author prefers this incision to any other for operative procedures on the cuff or inside the joint (see Chap 11, pp 386-387).

The interval between the deltoid and the

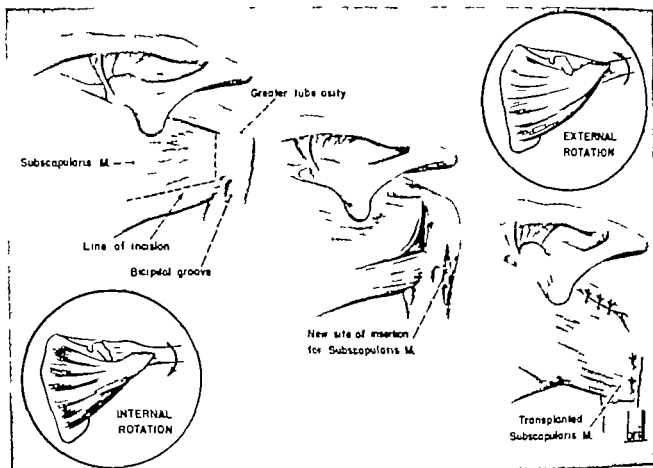


FIG 229 Magnuson operation (modified) for recurrent dislocation. The end of the subscapularis tendon is transferred to a bony trough below the greater tuberosity and parallel to the posterior lip of the bicipital groove.

the range of external rotation is decreased—which feature is obviously responsible for the satisfactory results.

Magnuson conceived a procedure whose simplicity and effectiveness are being recognized by many workers. By transferring the subscapularis tendon from the lesser to the greater tuberosity the range of external rotation is diminished; hence redislocation does not occur.

Discussion. A critical analysis of the many operative procedures devised for re-dislocation reveals that the essential feature which effects a cure is restriction of external rotation. Regardless of what region of the glenohumeral joint or at what components of the joint the operative attacks are aimed, limitation of external rotation will effect a cure in the great majority of cases. This is also nature's method of eliminating the disability. Therefore the simplest procedure

which will bring about restriction of the range of external rotation eventually will be adopted uniformly.

Magnuson points out that by the transference of the subscapularis tendon to the greater tuberosity a musculotendinous sling or cup is formed around the humeral head in both external and internal rotation which counterbalances the powerful pull of the adductor muscles (pectoralis major, latissimus dorsi, and teres major) which in turn tend to force the head downward and forward. It is the author's opinion that the Magnuson procedure properly performed, will supplant all other operations.

The author has slightly modified the operation in order to increase the efficiency of the musculotendinous sling. This has been accomplished by transferring the subscapularis tendon across the bicipital groove and anchoring it at a slightly lower level than

prove to be exceedingly difficult, as the tendon and the capsule blend into one mass just proximal to its insertion into the lesser tuberosity. The interval can be developed

laterally only as far as this fusion area. The subscapularis tendon is divided about one half an inch from its bony insertion and is allowed to retract medially. The an

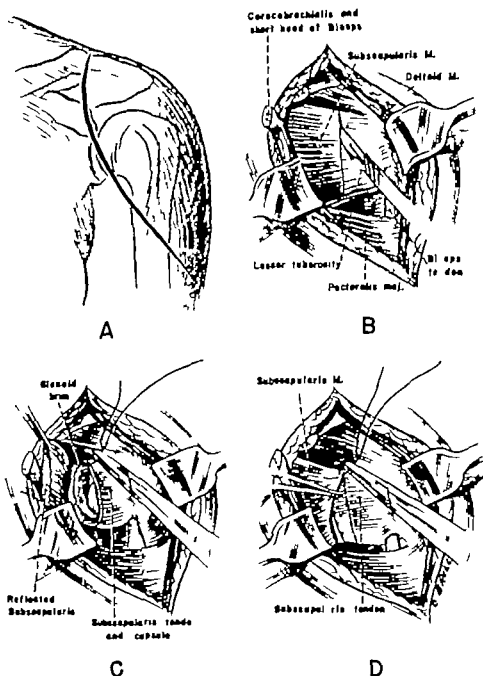


FIG. 231. Putti Platt Operation. (A) Anterior deltopectoral incision. (B) Conjoined tendon of the coracobrachialis and short head of the biceps tendon has been divided and retracted medially and downward. Subscapularis tendon is divided 1 inch from its insertion; the incision is also carried through the fibrous capsule. (C) With the arm rotated internally the lateral flap of the subscapularis muscle is sutured to the soft tissues on the anterior surface of the glenoid rim or to the labrum or occasionally to the under surface of the capsule and the subscapularis muscle. (D) The medial capsular flap is sutured to the outer surface of the lateral subscapularis tendon, thereby overlapping the first suture line. Finally the medial subscapularis flap is tacked to the tendinous portion of the cuff.

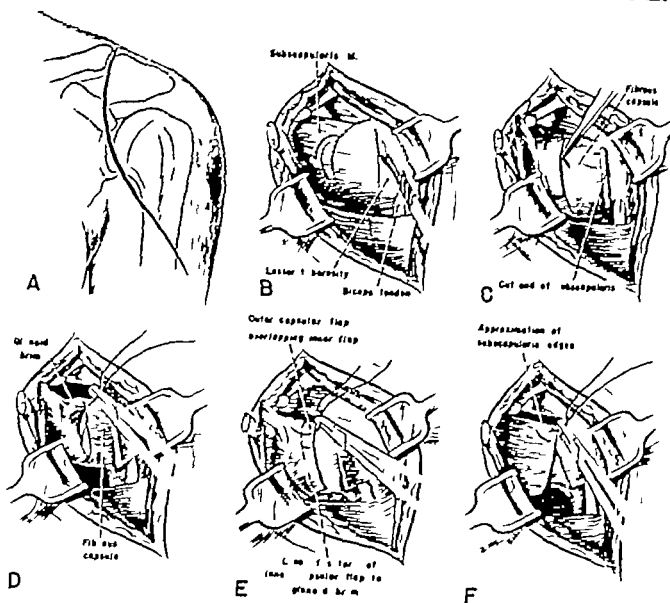


FIG. 230 Bankart Operation (A) Deltopectoral incision (an S-shaped skin incision may be substituted) (B) Coracoid process has been sectioned and retracted medially with the coracobrachialis and short head of the biceps tendon. The subscapularis tendon is sectioned $\frac{1}{2}$ inch from its bony insertion (C) The subscapularis muscle is peeled off the fibrous capsule and retracted medially. The capsule is opened by a 2 inch vertical incision 1 centimeter from the glenoid margin (D) four drill holes are made in the glenoid rim, and the lateral capsular flap is sutured to the bony margin of the glenoid fossa. (E) Medial capsular flap is overlapped and sutured to the lateral flap (F) Divided ends of the subscapularis tendon are approximated by interrupted silk or cotton sutures.

pectoralis major muscles is developed as far as the clavicle from which the deltoid muscle is detached partially and retracted laterally bringing into view the coracoid process and the tendons of the coracobrachialis the short head of the biceps and the pectoralis minor muscles. With an osteotome the coracoid process is sectioned and its tip is retracted downward and medially with the muscular attachment

External rotation of the humeral shaft brings into clear view the subscapularis tendon the lower border of which is marked by a plexus of veins. Division and ligation of these vessels provides an almost blood less field.

The interval between the subscapularis tendon and the capsule is developed medially as far back as the anterior glenoid margin. This part of the procedure may

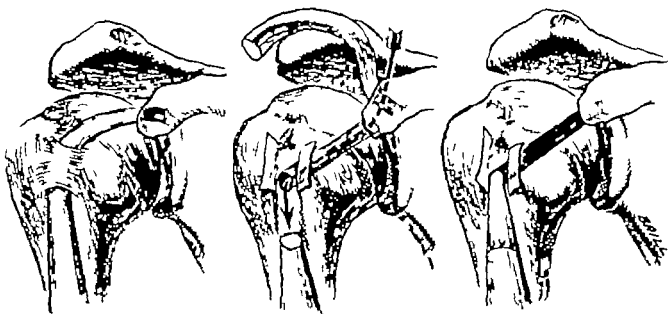


FIG. 233 The Nicola Operation. The biceps tendon is converted into a suspensory ligament for the head of the humerus.

tendon, thereby overlapping the first suture line. Further plication is provided by drawing the medial segment of the subscapularis muscle over the second suture line and anchoring it to the tendinous cuff over the greater tuberosity or the bicipital groove. Resuture of the conjoined tendon completes the operation. At this point in the procedure, external rotation should be limited to midposition of the arm. Closure of the wound in layers terminates the operation.

POSTOPERATIVE TREATMENT. After-care is similar to that recorded following the Bankart and the Magnuson operations.

Henderson Procedure. About 2 inches below the superior surface of the acromion process a curved skin incision is made from the anterior to the posterior aspect of the shoulder. The superior flap is reflected upward as far as the acromioclavicular joint, exposing the deltoid muscle and the acromion process. A drill hole (10 mm. in diameter) is made transversely through the acromion process. By blunt dissection a small opening is made in the anterior deltoid fibers and another in the posterior fibers exposing the anterior and the posterior surfaces of the greater tuberosity. A second transverse drill hole, paralleling the first, is made through the greater tuberosity. A segment of tendon 10 inches long

and half the thickness of the whole tendon is removed from the peroneus longus tendon of the leg on the affected side. It is passed through the drill holes and sutured to itself thereby forming an extracapsular suspensory ligament for the head of the humerus (Fig. 232).

POSTOPERATIVE TREATMENT. The arm is immobilized to the patient's side by a sling or swathe for 10 days. Active mobilization is then started, aided by physical therapy.

Nicola Procedure. The humeral head is exposed through an anterior incision beginning above the coracoid process and continued distally from 4 to 5 inches. The cephalic vein is identified and displaced medially with the pectoralis major muscle. By external rotation of the humerus, the intertubercular sulcus and the transverse humeral ligament are visualized. By division and retraction of the transverse humeral ligament the biceps tendon is seen within the bicipital groove. It is exposed further by dividing the capsule parallel with the fibers of the musculotendinous cuff. At the lower end of the bicipital groove two silk traction sutures are passed through the tendon which is then divided between the sutures. A traction suture on the distal end of the tendon ensures against its retraction down the arm. By means of a hand gauge

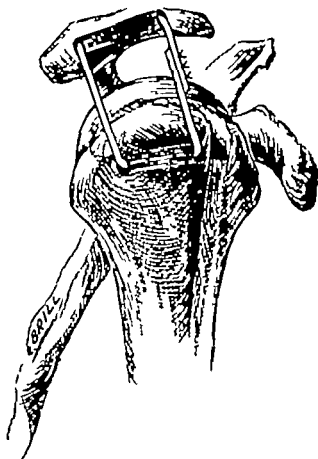


FIG. 232 Henderson's tenosuspension operation for recurrent dislocation.

terior margin of the glenoid rim and the capsule (which in this region forms the roof of the subscapularis recesses and is very thin) are identified readily. The interior of the joint is exposed by a 2 inch vertical incision approximately 1 cm from the glenoid margin hence creating a lateral and mesial capsular flap.

The anterior surface of the glenoid rim is roughened with a fine curette. Four drill holes are then made in the rim with a right angle dental drill. The lateral capsular flap is approximated and sutured to the bony rim. The mesial flap is lapped over the suture line and tacked to the lateral flap by interrupted sutures. Continuity of the subscapularis tendon is restored by resuturing its divided ends by interrupted silk sutures. Finally the tip of the coracoid process is sutured to the base and the wound is closed in layers.

POSTOPERATIVE TREATMENT This phase

of the treatment is identical with that described for the Magnuson operation.

Putti Platt Procedure (H. Osmond Clarke) This operation can be performed readily through a transacromial incision or through the anterior approach as described for the Magnuson and Bankart operations. Osmond Clarke utilizes an anterior incision extending inward along the outer third of the clavicle and then downward for approximately 6 inches. The incision curves along the medial margin of the coracoid process. The conjoined tendon (the coracobrachialis and the short head of the biceps brachii) is isolated and divided from the coracoid process, care being taken to leave a proximal stump sufficiently long to facilitate subsequent resuture. By gentle retraction the conjoined tendon is displaced downward and medially. However, care must be taken to avoid injury to the musculocutaneous nerve or the axillary vessels and nerves which run along the medial border of the coracobrachialis muscle.

External rotation of the shaft brings into view the subscapularis muscle and tendon, its lower border being identified by a plexus of veins which are carefully divided and tied. The tendon is elevated by a blunt sector passing beneath it from above or below and then is divided one inch from its insertion. This incision also divides the fibrous capsule thereby opening the joint cavity and providing visualization of the interior of the glenohumeral joint.

While holding the arm internally rotated the lateral flap of the subscapularis muscle is sutured to the soft tissues on the anterior surface of the glenoid rim or to the labrum. It may be necessary to suture the tendon to the undersurface of the capsule and the subscapularis muscle.

In such instances the anterior surface of the neck of the scapula is scarified by a fine osteotome. Four interrupted silk sutures suffice to anchor the lateral end of the muscle to its new bed. The medial flap of the capsule is then tacked to the outer surface of the lateral portion of the subscapularis

POSTERIOR RECURRENT DISLOCATION

This is a rare lesion, but it may follow a primary traumatic dislocation or occur spontaneously without history of injury. Frequently it is bilateral. Most observers make a distinction between recurrent and habitual dislocation.

Recurrent dislocation usually follows a violent injury which produces a posterior dislocation. It tends to recur at irregular intervals when the arm is subjected unduly to specific stresses.

Habitual dislocation may result from an acute primary injury, but more frequently it occurs spontaneously without history of trauma, and in many instances it is bilateral. The lesion occurs every time the arm is elevated in the coronal or frontal plane, particularly if it is raised in internal rotation. A recurrent lesion may become habitual.

PATHOLOGY

The pathologic findings essentially are those recorded for anterior dislocation. Stripping of the capsule and the labrum from the posterior glenoid rim has been observed in some cases. In others the labral and the capsular attachments are intact, but the capsule is stretched and pouches out

under the external rotators. The latter condition is more frequently encountered in the habitual luxations. Regardless of the local abnormalities there is loss of normal tonicity of the rotator muscles, particularly the external group. This is evidenced by their laxity.

Again, it becomes apparent that the local changes are not responsible for recurrences, but rather that the true etiologic factor is a neuromuscular in-co-ordination or imbalance which may be inherent, as in the bilateral habitual luxations (which occasionally are observed in more than one member of the same family). Again it may be the sequel of violent trauma to the local neuromuscular apparatus sustained at the initial dislocation.

TREATMENT

The operative procedures which have been described for anterior dislocations have been employed for posterior lesions also. In the light of the author's concept of the causative factors, the local alterations are not disturbed. Three cases have been cured by simple advancement of the insertion of the *infraspinatus* and *teres minor* tendons with the arm in external rotation. This procedure restricts internal rotation but increases the efficiency of the external rotators thereby preventing recurrences.

BIBLIOGRAPHY

- Adams C. J. Recurrent dislocation of the shoulder. *J. Bone & Joint Surg.* 30 B 26-38 1948.
- Allen, A. W. Living suture grafts in repair of fractures and dislocations. *Arch. Surg.* 16 1007 1938.
- Bankart A. S. Recurrent or habitual dislocation of the shoulder joint. *Brit. M. J.* 2 1132 1923.
- Bankart A. S. The pathology and treatment of recurrent dislocation of the shoulder joint, *Brit. J. Surg.* 26 23 1938.
- Bankart A. S. An operation for recurrent dislocation (subluxation) of the sternoclavicular joint. *Brit. J. Surg.* 26 320 1938.
- Bennett G. E. Old dislocations of the shoulder. *J. Bone & Joint Surg.* 18 594-606 1936.
- Bost F. C., and Laman V. T. The pathological changes in recurrent dislocation of the shoulder. *J. Bone & Joint Surg.* 24 595-613 1942.
- Bosworth, B. M. Acromioclavicular separation: a new method of repair. *Surg. Gynec. & Obst.* 73 866-871 1941.
- Bunnell Sterling. Fascial grafts for dislocation of acromioclavicular joint. *Surg. Gynec. & Obst.* 46 563-564 1928.
- Burrell, H. L., and Lovett R. W. Habitual or Recurrent Dislocation of the Shoulder. *Am. J. M. Sc.* 114 166 1897.
- Cadenat F. M. Treatment of dislocations and fracture of the outer end of the clavicle. *Internat. Clin., Series* 27 1 145 169 1917.

TABLE 1
ANALYSIS OF 23 MAGNUSON PROCEDURES (MODIFIED)

Name	Sex	Age	Date of Operation	Number of Recurrences	Pain	Satisfied
C F	M	28	5-26-48	1	Yes	No
R M	M	23	11-22-47	0	No	Yes
R C	M	30	5-30-48	0	No	Yes
G H	M	24	10-3-47	0	Occasionally	Yes
W M	M	24	6-28-48	0	No	Yes
A A	M	23	2-30-48	0	No	Yes
M K	M	21	3-21-48	0	No	Yes
F H	M	22	4-29-48	0	No	Yes
J C	M	29	6-25-48	0	No	Yes
J F	M	26	7-30-48	0	No	Yes
W A	M	20	7-19-48	0	Yes	Yes
A A	M	25	8-24-48	6	After Dislocation	No
W S	M	21	3-3-48	0	No	Yes
J N	M	18	11-15-47	0	No	Yes
T J	M	36	8-4-48	0	No	Yes
D L	M	17	5-21-45	0	No	Yes
W B	M	30	1-21-48	0	No	Yes
C M	F	29	3-26-48	0	Slight	Yes
J C	M	19	8-24-47	0	No	Yes
L B	M	33	6-12-47	0	No	Yes
T N	M	22	8-18-46	0	No	Yes
D O	M	29	10-15-47	0	No	Yes
J N	M	22	2-23-48	0	No	Yes

Analysis of Cases

Total number of cases	23
Age range	17 to 33 years
Males	22
Females	1
Recurrences of dislocation	2 (8.7 per cent)
Pain present	2
Not satisfied with operation	2
Satisfied with operation	21
Period of observation	17 mo. to 3½ yr

a quarter inch tunnel is made beginning at the lower end of the bicipital groove and emerging out of the center of the articulating surface of the head of the humerus. Nicola stresses that the exit of the tunnel be in the center of the head of the humerus.

The proximal segment of the biceps tendon is stripped of its synovial sheath and then by the use of a wire loop its traction suture is passed through the tunnel from above downward. Steady traction on the silk suture pulls the tendon through. Continuity is restored by suturing the proximal and the distal ends of the tendon which in

turn is anchored through drill holes to the transverse humeral ligament onto the sides of the groove. Closure of the capsule is effected by side-to-side sutures of silk. The operation is completed by closure of the wound in layers.

POSTOPERATIVE TREATMENT The arm is immobilized to the side for 4 weeks by means of a Velpeau dressing. Progressive, active stooping exercises are then begun. Physical therapy is a valuable adjunct in that properly utilized it impresses the patient with the importance of active progressive movements necessary for complete restoration of function.

POSTERIOR RECURRENT DISLOCATION

This is a rare lesion, but it may follow a primary traumatic dislocation or occur spontaneously without history of injury. Frequently it is bilateral. Most observers make a distinction between recurrent and habitual dislocation.

Recurrent dislocation usually follows a violent injury which produces a posterior dislocation. It tends to recur at irregular intervals when the arm is subjected unduly to specific stresses.

Habitual dislocation may result from an acute primary injury, but more frequently it occurs spontaneously without history of trauma, and in many instances it is bilateral. The lesion occurs every time the arm is elevated in the coronal or frontal plane, particularly if it is raised in internal rotation. A recurrent lesion may become habitual.

PATHOLOGY

The pathologic findings essentially are those recorded for anterior dislocation. Stripping of the capsule and the labrum from the posterior glenoid rim has been observed in some cases. In others the labral and the capsular attachments are intact, but the capsule is stretched and pouches out

under the external rotators. The latter condition is more frequently encountered in the habitual luxations. Regardless of the local abnormalities, there is loss of normal tonicity of the rotator muscles, particularly the external group. This is evidenced by their laxity.

Again, it becomes apparent that the local changes are not responsible for recurrences, but rather that the true etiologic factor is a neuromuscular in-co-ordination or imbalance which may be inherent, as in the bilateral habitual luxations (which occasionally are observed in more than one member of the same family). Again it may be the sequel of violent trauma to the local neuromuscular apparatus sustained at the initial dislocation.

TREATMENT

The operative procedures which have been described for anterior dislocations have been employed for posterior lesions also. In the light of the author's concept of the causative factors, the local alterations are not disturbed. Three cases have been cured by simple advancement of the insertion of the *infraspinatus* and *teres minor* tendons with the arm in external rotation. This procedure restricts internal rotation but increases the efficiency of the external rotators, thereby preventing recurrences.

BIBLIOGRAPHY

- Adams, C. J. Recurrent dislocation of the shoulder. *J. Bone & Joint Surg.* 30-B:26-38, 1948.
- Allen, A. W. Living suture grafts in repair of fractures and dislocations. *Arch. Surg.* 16:1007, 1938.
- Bankart, A. S. Recurrent or habitual dislocation of the shoulder joint. *Brit. M. J.* 2:1132, 1923.
- Bankart, A. S. The pathology and treatment of recurrent dislocation of the shoulder joint. *Brit. J. Surg.* 26:23, 1938.
- Bankart, A. S. An operation for recurrent dislocation (subluxation) of the sternoclavicular joint. *Brit. J. Surg.* 26:320, 1938.
- Bennett, G. E. Old dislocations of the shoulder. *J. Bone & Joint Surg.* 18:594-606, 1936.
- Bost, F. C. and Inman, V. T. The pathological changes in recurrent dislocation of the shoulder. *J. Bone & Joint Surg.* 24:595-613, 1942.
- Bosworth, B. M. Acromioclavicular separation: a new method of repair. *Surg. Gynec. & Obst.* 73:866-871, 1941.
- Bunnell, Sterling. Fascial grafts for dislocation of acromioclavicular joint. *Surg. Gynec. & Obst.* 46:563-564, 1928.
- Burrell, H. L., and Lovett, R. W. Habitual or Recurrent Dislocation of the Shoulder. *Am. J. M. Sc.* 114:166, 1897.
- Cadenat, F. M. Treatment of dislocations and fracture of the outer end of the clavicle. *Internat. Clin., Series* 27:1:145-169, 1917.

- Codman E. A. *The Shoulder* Boston Thomas Todd, 1934
- Cubbins, W. R., Callahan J. S. and Scuder C. S. Irreducible shoulder dislocations *Surg., Gynec & Obst* 58 129 1934
- Davis A. G. A conservative treatment for habitual dislocation of the shoulder *J.A.M.A.* 107 1012 1015 1936.
- Delbet P. Des luxations anciennes et irreductibles de l'épaule *Arch. gen. de med.* 1 144 1893
- Dessaint J. J. Sur l'adaptation fonctionnelle dans les luxations anciennes antero-intermes de l'épaule *Rev. d'orthop.* 15 508 1928
- Duchenne G. B. *Physiologie des mouvements* Paris Baillière 1867 pp. 17 (Tr B Kaplan Philadelphia, Lippincott 1949)
- Evre-Brook A. L. Recurrent dislocation of the shoulder *J Bone & Joint Surg* 30 B 39-46 1948.
- Gallie W. E. Dislocations *New England J Med.* 213 91 1935
- Gallie W. E. and LeMesurier A. B. An operation for the relief of recurring dislocation of the shoulder *Trans. Am. Surg. Assn* 45 392 1927
- Gallie W. E., and LeMesurier A. B. Recurring dislocation of the shoulder *J Bone & Joint Surg* 30 B 9 18 1948
- Groves E. W. Hey Recurrent dislocation of the shoulder a plea for a simple operation *Brit J Surg* 26 3/5 1938
- Gurd, F. B. The treatment of complete dislocations of the outer end of the clavicle *Ann. Surg* 113 1094 1098 1941
- Hendenach, J. C. R. Recurrent posterior dislocation of the shoulder *J Bone & Joint Surg* 29 582 586 1947
- Mumford, E. B. Acromioclavicular dislocation a new operative treatment *J Bone & Joint Surg* 23 799-802 1941
- Murray C. R. Dislocation of the shoulder *J.A.M.A.* 96 337 1931
- Neviaser J. S. Operation for old dislocation of the shoulder *J Bone & Joint Surg* 30-A 99 1000 1948
- Nicola T. Recurrent anterior dislocations of the shoulder a new operation *J Bone & Joint Surg* 11 128 1929
- Nicola T. Recurrent dislocation of the shoulder its treatment by transplantation of the long head of the biceps *Am J Surg* 6 815 1929
- Nicola, T. Recurrent dislocation of the shoulder *J Bone & Joint Surg* 16 663 1934
- Nicola, T. Anterior dislocation of the shoulder the role of the articular capsule *J Bone & Joint Surg* 24 614-616 1942
- Nicola, T. Acute anterior dislocation of the shoulder *J Bone & Joint Surg* 31 A 153 159 1949
- Osmond-Clarke, H. Habitual dislocation of the shoulder *J Bone & Joint Surg.* 308 19-25 1948
- Oudard M. La luxation recidivante de l'épaule (variété antero-interne) procédé opératoire, *J de chir* 23 13 25 1923 [Abst. *J.A.M.A.* 82 1004 1924]
- Palmer L., and Widen A. Bone block method for recurrent dislocation of the shoulder joint, *J Bone & Joint Surg* 308 53-58 1948
- Perthes G. Über Operationen bei Habituellem Schulterluxation *Deutsche Ztschr f Chir* 85 199 1906
- Pthemster D. B. The treatment of dislocations of the acromioclavicular joint by open reduction and threaded wire fixation, *J Bone & Joint Surg* 24 166-168 1942
- Roberts S. M. Acromioclavicular dislocation, *Am. J Surg* 23 322 1934
- Robertson R., and Stark, W. J. Diagnosis and treatment of recurrent dislocation of the shoulder *J Bone & Joint Surg* 20 97 800 1947
- Rowe C. R. and Yee L. B. A posterior approach to the shoulder joint *J Bone & Joint Surg* 26 580-584 1944
- Schneider C. C. Acromioclavicular dislocation, *J Bone & Joint Surg.* 15 95 1933
- Schwartz, E. O. P. Die habituellen schulter Luxation klinischer und experimenteller Beitrag, *Arch. f klin. Chir* 104 138 1914
- Speed, J. S., and Smith, H. *Campbell's Operative Orthopedics* ed. 2 vol 2 p. 347. St. Louis, Mosby 1949
- Speed, K. Recurrent anterior dislocation of the shoulder operative cure by bone graft *Surg. Gynec & Obst* 44 468 1927
- Stevens J. H. The action of the short rotators on the normal abduction of the arm with a consideration of their action in some cases of subacromial bursitis and allied conditions *Am. J M Sc* 138 8 0-834 1909
- Stevens J. H. Dislocation of the shoulder *Ann. Surg* 83 84-103 1926
- Stimson L. A. *A Practical Treatise on Fractures and Dislocations* ed. 4 p 59 Philadelphia Lea 1905
- Thomas M. A. Posterior subacromial dislocations of the head of the humerus. *Am. J Roentgenol.* 27 67, 3 1937
- Thomas T. T. Habitual or recurrent dislocation of the shoulder etiology pathology and treatment *Am J M Sc* 138 229-367 1909
- Thomas T. T. Recurrent anterior dislocation of the shoulder with a report of three cases treated by capsulorrhaphy through an axillary incision, *J A M A.* 54 834 1910.
- Thomas T. T. Habitual or recurrent dislocation

- of the shoulder eighteen shoulders operated on in sixteen patients—a new axillary Surg., Gynec & Obst. 18 10/ 1914
- Thomas, T T Recurrent dislocation of the shoulder joint J.A.M.A. 85 1202 1925
- Unst M R. Complete dislocation of the acromioclavicular joint the nature of the traumatic lesion, and effective methods of treatment with an analysis of forty-one cases J Bone & Joint Surg. 28 313 1946
- Vargas Luthero Repair of complete acromioclavicular dislocation utilizing the short head of the biceps J Bone & Joint Surg 24 772 ,73 1942
- Vere-Hodge Quoted by Watson Jones in Fractures and Joint Injuries ed. 3 vol. 2 p. 434 Baltimore Williams & Wilkins 1946
- Watkins J T Operation for acromioclavicular luxations J Bone & Joint Surg 7 790 1925
- Watson Jones R. Fracture and Joint Injuries ed. 3 vol 2, pp 433-435 Williams & Wilkins 1946
- Wilson J C and McKeever F M Traumatic posterior dislocation of the humerus J Bone & Joint Surg. 31 A 160-172 1949

8

Fractures and Fracture-Dislocations of the Upper End of the Humerus

GENERAL CONSIDERATIONS

MECHANISM OF FRACTURES

CLASSIFICATION OF FRACTURES

FRACTURES REQUIRING NO REDUCTION

FRACTURES REQUIRING REDUCTION

SEPARATION OF THE HUMERAL EPIPHYSIS

FRACTURE-DISLOCATION OF THE HUMERUS

FRACTURES OF THE GREATER TUBEROSITY

FRACTURES OF THE HEAD AND NECK OF THE HUMERUS, WITH DISLOCATION

GENERAL CONSIDERATIONS

Optimum management of fractures of the upper humerus is governed by the knowledge of certain anatomic and clinical features peculiar to this region. Prime consideration must be given to the patient as a whole. The local pathology is of secondary importance. The individual's age, sex, occupation and economic plane must be adequately evaluated, since these factors have a direct bearing upon the preferred method of treatment.

The ideal objectives in the treatment of the above lesions are (1) normal anatomic restoration of all the involved tissues (osseous and soft tissues) and (2) complete functional return of the extremity and the individual in the shortest period of time possible. Clinical experience, however, makes it clear that both objectives cannot always be achieved and that the first is not essential to achieve the second. One must also appreciate that this principle is applicable only within certain limits and if the limits are exceeded, poor function and pronounced disability are inevitable.

Features peculiar to the shoulder region are:

1. Stability of the glenohumeral joint is not dependent upon the configuration of its bony components, but upon the muscles

which motorize it, primarily those comprising the musculotendinous cuff. Incongruity of the glenohumeral surfaces, within reasonable limits, is compatible with good function, because the muscular apparatus can adjust itself to changes in the architecture of the bony elements. In the light of such facts, one is justified in accepting deformities of the upper end of the humerus following injury, provided that the malalignment can be compensated for by the soft tissue structures. This is particularly true of all impacted fractures of the upper end of the humerus.

2. Motion in the glenohumeral joint is the result of a group of muscles working as a unit, but failure of one muscle of the group to function, regardless of the cause, does not materially affect the overall performance of the unit. This again is true only within certain limits. For example, perfect shoulder function is attained in cases with complete tears in the musculotendinous cuff, provided that the defects are not extensive. If there is severe disruption of the motor apparatus, marked loss of function is the rule unless the lesion is recognized and adequate repair effected.

3. Bony union is assured and not influenced by any method of treatment. Impacted fractures always heal readily. Unun-

pacted fractures heal, provided that the fragments maintain some contact. Non union occurs only in fractures with marked displacement of the fragments, as occur in fracture dislocations in which the head lies in the soft tissues at considerable distance from the distal fragment and separated from it by soft tissue.

4 The intimate relation of the insertion of the rotator muscles into the tuberosities tends to preserve continuity of fragments in fractures involving this region. Continuity is also enhanced by the subacromial bursa which is closely adherent to the superior surfaces of the tuberosities, the periosteum which may be intact at the lower borders of the fragments and to some degree by the biceps tendon which tends to maintain normal anatomic relationship of the fragments.

There are exceptions to these observations. Fractures through the anatomic neck may result in complete displacement of the humeral head, the degree of displacement depending upon the violence of the injury (the head may be forced out of the capsule). Fractures of the tuberosities with retraction of the fragments denote extensive tearing of the musculotendinous cuffs.

In the light of these observations, it becomes obvious that in impacted fractures maintenance of the position of the fragments can be expected without the use of cumbersome apparatus, splints or casts which are superfluous and may be harmful.

5 Preservation of the gliding mechanism between the different soft tissue strata of the shoulder is essential for rapid and complete restoration of function. Traumatized soft tissue in this region tends to heal rapidly. Secondary fibrosis, contraction and loss of elasticity are common sequelae of this reparative process. Early mobilization within safe limits (within the tolerance of pain) is the most effective therapeutic agent available to prevent stiffness and to preserve free movement. Early mobilization should be the most important feature of the therapeutic regimen in patients over 40 years of

age and in the aged in whom there are already varying degrees of degenerative changes in the supportive soft tissue structure resulting from wear and tear and senescence, because such tissues are more likely to develop protracted stiffness than the tissues of patients under 30.

MECHANISM OF FRACTURES OF THE UPPER END OF THE HUMERUS

Direct violence to the shoulder rarely produces fractures of the upper end of the humerus. The overhanging acromion protects the humeral head from the impact of direct forces. Trauma of this nature, if of sufficient severity, usually results in either a fracture of the clavicle or disruption of the acromioclavicular joint. In rare instances direct trauma to the point of the shoulder with the arm in dorsiflexion may produce a fracture of the upper end of the humerus.

Fractures of this region are produced in variably by the same mechanism that is responsible for dislocation of the humeral head. They result from such indirect violence as falls on the outstretched arm, during which the arm fails to rotate quickly enough in the right plane to reach the safety of the pivotal position, or it is forced beyond the limits of this enclosure. The tough acromion process acts as a fulcrum at the base of the greater tuberosity, while the shaft of the humerus becomes the long arm of the lever. As the arm is forced into hyperabduction the superior rim of the glenoid fossa is wedged between the greater tuberosity and the articular head. If the force continues, either a dislocation or a fracture of the humerus or a combination of both results. Codman recognized a false and a true dislocation.

In a false dislocation the head is levered out of the glenoid cavity without rupture of the inferior capsule which becomes unduly stretched to accommodate the heads. Spontaneous reduction occurs as the arm descends to the side from the abducted posi-

tion In true dislocation the head is forced through a rent in the capsule and remains in a subglenoid position after the arm is brought to the side Both forms may be accompanied by fractures of the upper end of the humerus

In general the above mechanism is responsible for different lesions in the various age groups In children the weakest point in the upper end of the humerus is its epiphyseal line therefore epiphyseal separation is the most common lesion encountered In youth and manhood after the epiphyseal lines have been obliterated, the osseous and tendinous elements are the strongest tissues in this region On the other hand the capsule is relatively weak and dislocations are more likely to occur If a fracture does occur it is as a rule through the base of the greater tuberosity or the surgical neck Finally in the old age periods the cancellous bone is more or less brittle and weak hence comminution of the upper end of the humerus is a frequent occurrence

Other factors which influence the type of lesion produced by the described mechanism are

1 The severity of the force and the exact point on the humerus at which it is expended The acromion will act as a fulcrum at different points on the tuberosity, depending upon the degree of rotation of the humeral shaft Appreciation of this observation makes it apparent that numerous combinations of fracture lines through this region are possible

2 The weight and the velocity of the falling body

3 The direction the arm takes upon striking the ground before the full impact of the disruptive force is expended If it moves toward the body, an adduction fracture results If it moves away from the body an abduction fracture is produced (Fig 234) In adults the abduction type is more common while in children the adduction form is more frequently encountered the abduction fracture being exceedingly rare (Fig 235)



FIG 234 (Left) Abduction fracture of the neck of the humerus, resulting from a fall on the outstretched arm the arm moving away from the body

FIG 235 (Right) Adduction fracture of the neck of the humerus, resulting from a fall on the outstretched arm the arm moving toward the body

Comminuted fractures of the upper end of the humerus, as previously recorded usually occur in elderly individuals but may occur in any age group. After closure of the epiphyses a thin plate of hard bone replaces the epiphyseal lines. These partitioning bony

plates are readily discernible in humeral heads of the aged. They consist of firmer, tougher, more compact bone than the adjacent cancellous bone. Codman pointed out that the lines of cleavage of most fractures in this region follow near these old lines of



FIG 236 A disruptive force produced by impingement of the humerus against the acromion (insert) may produce fractures following the planes of the lines of epiphyseal union resulting in 4 main fragments or any combination of these fragments.

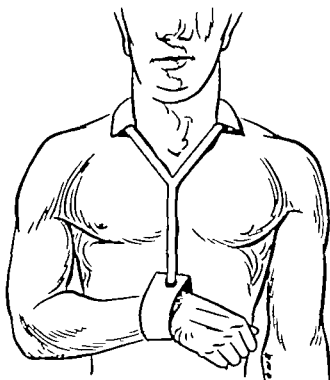


FIG 237 Collar and-cuff sling

epiphyseal union and the head of the bone tends to become divided into four main fragments or various combinations of these four fragments (Fig 236)

CLASSIFICATION OF FRACTURES OF THE UPPER END OF THE HUMERUS

Analysis of the various types of fractures which may be possible by the aforementioned mechanism permits the following classification from an anatomic viewpoint

- 1 Fracture of the greater tuberosity
- 2 Fracture of the lesser tuberosity
- 3 Fracture of the anatomic neck
- 4 Fracture of the surgical neck
- 5 Separation of the epiphysis of the humerus
- 6 Fracture of the head of the humerus
- 7 Any combination of the above fractures.
- 8 Comminuted fractures of the entire upper end of the humerus
- 9 Fracture-dislocations Any of the above fractures may occur with dislocation of the humeral head

Although such a classification denotes to some degree the topographic anatomy of the

fracture, it is of no value from a therapeutic viewpoint. The author is in complete agreement with McLaughlin in that fractures of the upper end of the humerus should be divided into two principal groups

- 1 Fractures requiring no reduction
- 2 Fractures requiring reduction.

FRACTURES REQUIRING NO REDUCTION

GENERAL CONSIDERATIONS

The great majority of fractures involving the upper end of the humerus needs no reduction. This is particularly true of all impacted fractures and includes fractures of the surgical neck, the anatomic neck, tuberosities, or any combination of the above. Only unimpacted fractures which demonstrate gross disalignment and angulation and surgical and anatomic neck fractures with complete displacement demand repositioning of the fragments. As previously stated certain functional and anatomic features peculiar to this region justify this method of treatment. They are

- 1 Anatomic repositioning of fragments

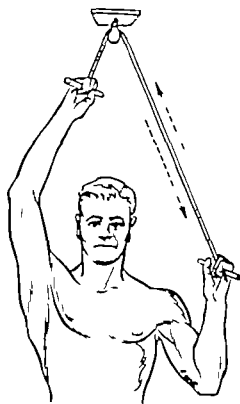


FIG 238 Exercises for shoulder

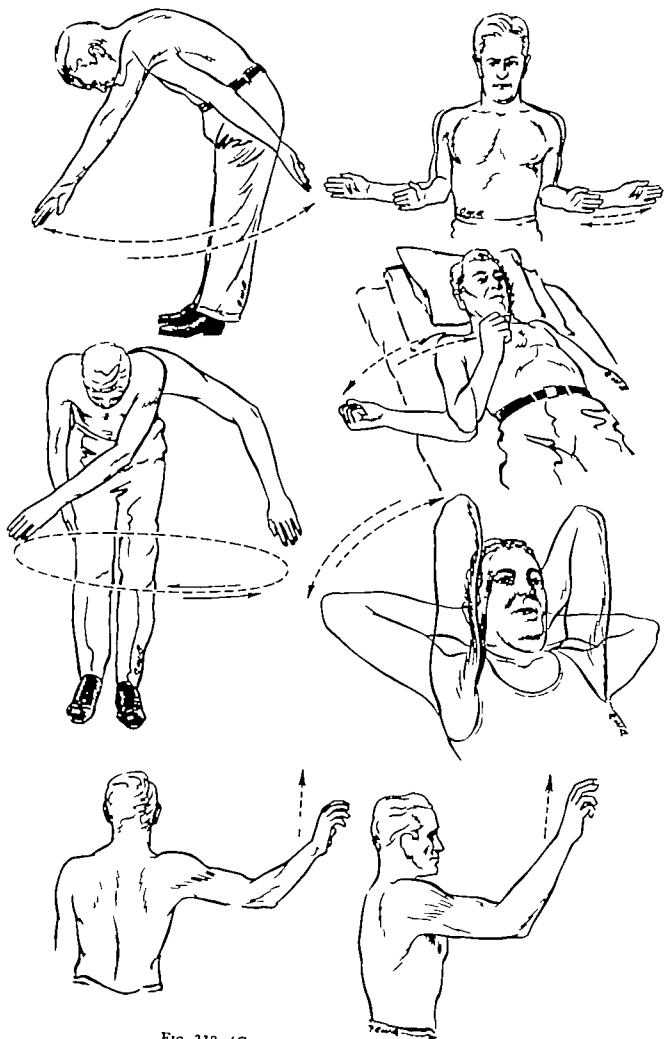


FIG 238 (Continued) Exercises for shoulder

is not essential for normal function. Clinical experience points out that incongruity of the articular surfaces of the glenohumeral joint is compatible with good function.

2 Further displacement of the fragments following the initial violence does not occur, because the musculotendinous cuff, the subacromial bursa and the biceps tend to keep the fragments together.

3 Bone healing always occurs except in instances of wide separation of the fragments with interposition of soft tissues.

4 Changes in the mechanics of the muscular apparatus within reasonable limits do not preclude good function. This is particularly true of the insertion of the short rotators into the head of the humerus.

5 Early mobilization of the glenohumeral joint within the limits of pain does not displace the fragments (cf *supra* 2) favors early restoration of function and prevents stiffness by preserving the gliding mechanism between the various soft tissue strata of the shoulder.

MANAGEMENT OF IMPACTED FRACTURES OF THE UPPER END OF THE HUMERUS

All cumbersome fixation apparatus such as abduction plaster casts, hanging casts, splints and braces are definitely contraindicated in the management of this group of fractures. A simple sling and swathe holding the arm to the side suffices. It allows early motion and the weight of the arm provides adequate traction at the fracture site. Even better is a collar-cuff sling suspending the arm from the neck (Fig 237).

Motion in impacted fractures is started immediately by encouraging the patient to perform stooping exercises (Fig 238). All movements must be within the patient's tolerance of pain—never beyond.

If the regimen is carried out properly, exercises are done a specific number of times (from 8 to 10 times) every hour or for a specified period of time (from 3 to 5 minutes) every hour. The range of motion and the period of the exercises increases progressively as the intensity of the pain

subsides. After 3 weeks, the sling may be discarded during the day. The swathe is worn only at night to protect the extremity from involuntary movements. Two or three days after the injury, wall-crawling and later, pulley exercises are added. Free use of the arm within the limits of pain is encouraged at all times, while physical therapy in the form of radiant heat and gentle massage is a valuable adjunct to the treatment. Maximum restoration of function should be achieved in from 8 to 12 weeks.

Motion in unimpacted fractures should be performed with greater caution than is sometimes exercised. Movements against gravity should not be attempted until there is clinical evidence of sufficient healing to preclude further displacement of the fragments. This is manifest by all fragments moving in unison upon gentle internal and external rotation of the humeral shaft. Bony healing sufficient to permit antigravity exercises usually is attained in 2 or 3 weeks.

The above regimen must be adhered to religiously in patients past 40 years of age. Only by such a program the principal feature of which is early mobilization can protracted stiffness and great disability be averted.

In general, the prognosis in fractures of this group treated by the afore-mentioned method is good. However, there are two fractures in this category whose prognosis, regardless of the treatment, is invariably poor. These fractures are (1) depressed fractures of the greater tuberosity and (2) impacted fractures of the anatomic head into greater tuberosity.

DEPRESSED FRACTURE OF THE GREATER TUBEROSITY

As a rule, it is produced by a direct force on the point of the shoulder when the arm is in dorsal flexion, or by impingement of the greater tuberosity against the acromion process, but it may be the result of a telescoping force driving the greater tuberosity against the glenoid during falls with the arm in forward elevation or abduction.

Invariably, this injury is complicated by pronounced periosteal injury, extensive injury to the insertion of the musculotendinous cuff, and distortion of the normal configuration of the bicipital groove. Because

sides and the floor of the groove, and gross obliteration of portions of the sulcus are common sequelae to this form of fracture. Treatment of depressed fractures of the greater tuberosity is similar to that de-



FIG. 239 Impacted depressed fractures of the greater tuberosity. Stiff and painful shoulders are frequent undesirable sequelae of this type of fracture. The painful syndrome usually results from a bicipital tenosynovitis, which may be primary or secondary in origin. Such fractures often implicate the intertubercular sulcus distorting its configuration.

of extensive periosteal damage, the fracture may be complicated by massive callus formation which impinges against the coracoacromial ligament or the acromion on elevation of the arm.

Distortion of the bicipital groove may interfere with the normal tendon-tendon sheath gliding mechanism of the biceps tendon, resulting in a bicipital tenosynovitis (Fig. 239, right). In elderly patients, this lesion may be responsible for the development of frozen shoulder. This crippling protracted complication is initiated by a bicipital tenosynovitis which results from trauma sustained at the time of the initial violence or develops gradually following secondary osseous changes in the bicipital groove. Roughening, spur formation on the

scribed for impacted fractures. Disabling sequelae should be anticipated and treated as soon as they manifest themselves. The prognosis always should be a guarded one.

IMPACTED FRACTURES OF THE ANATOMIC HEAD INTO GREATER TUBEROSITY

This lesion is encountered more frequently since the advent of shock therapy. The author has seen three instances following convulsive seizures produced by this form of treatment. The anatomic head is driven into the cancellous bone of the tuberosity, cutting deeply into the musculotendinous cuff at its line of insertion into the sulcus. The intertubercular groove is obliterated, thereby crushing the biceps tendon and obliterating its gliding mechanism.

is not essential for normal function. Clinical experience points out that incongruity of the articular surfaces of the glenohumeral joint is compatible with good function.

2. Further displacement of the fragments following the initial violence does not occur, because the musculotendinous cuff, the subacromial bursa and the biceps tend to keep the fragments together.

3. Bone healing always occurs except in instances of wide separation of the fragments with interposition of soft tissues.

4. Changes in the mechanics of the muscular apparatus within reasonable limits do not preclude good function. This is particularly true of the insertion of the short rotators into the head of the humerus.

5. Early mobilization of the glenohumeral joint within the limits of pain does not displace the fragments (cf *supra* 2) favors early restoration of function and prevents stiffness by preserving the guiding mechanism between the various soft tissue strata of the shoulder.

MANAGEMENT OF IMPACTED FRACTURES OF THE UPPER END OF THE HUMERUS

All cumbersome fixation apparatus such as abduction plaster casts, hanging casts, splints and braces are definitely contraindicated in the management of this group of fractures. A simple sling and swathe holding the arm to the side suffices. It allows early motion and the weight of the arm provides adequate traction at the fracture site. Even better is a collar-cuff sling suspending the arm from the neck (Fig. 237).

Motion in impacted fractures is started immediately by encouraging the patient to perform stooping exercises (Fig. 238). All movements must be within the patient's tolerance of pain—never beyond.

If the regimen is carried out properly exercises are done a specific number of times (from 8 to 10 times) every hour or for a specified period of time (from 3 to 5 minutes) every hour. The range of motion and the period of the exercises increases progressively as the intensity of the pain

subsides. After 3 weeks, the sling may be discarded during the day. The swathe is worn only at night to protect the extremity from involuntary movements. Two or three days after the injury, wall-crawling and later, pulley exercises are added. Free use of the arm within the limits of pain is encouraged at all times, while physical therapy in the form of radiant heat and gentle massage is a valuable adjunct to the treatment. Maximum restoration of function should be achieved in from 8 to 12 weeks.

Motion in unimpacted fractures should be performed with greater caution than is sometimes exercised. Movements against gravity should not be attempted until there is clinical evidence of sufficient healing to preclude further displacement of the fragments. This is manifest by all fragments moving in unison upon gentle internal and external rotation of the humeral shaft. Bony healing sufficient to permit antigravity exercises usually is attained in 2 or 3 weeks.

The above regimen must be adhered to religiously in patients past 40 years of age. Only by such a program, the principal feature of which is early mobilization, can protracted stiffness and great disability be averted.

In general the prognosis in fractures of this group treated by the afore-mentioned method is good. However there are two fractures in this category whose prognosis, regardless of the treatment, is invariably poor. These fractures are (1) depressed fractures of the greater tuberosity and (2) impacted fractures of the anatomic head into greater tuberosity.

DEPRESSED FRACTURE OF THE GREATER TUBEROSITY

As a rule it is produced by a direct force on the point of the shoulder when the arm is in dorsal flexion or by impingement of the greater tuberosity against the acromion process, but it may be the result of a telescoping force driving the greater tuberosity against the glenoid during falls with the arm in forward elevation or abduction.

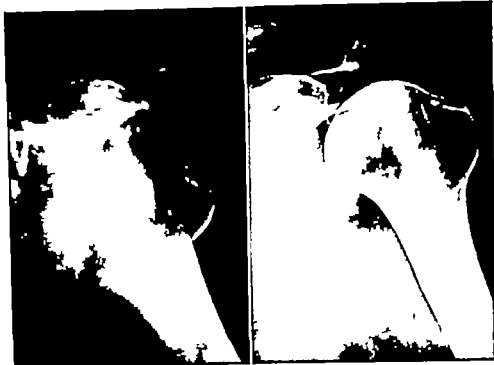


FIG 242 (*Left*) Fracture of the greater tuberosity associated with anterior dislocation of the head of the humerus. Note that the acromion has retained its normal position relative to the scapula. (*Right*) Fracture-dislocation depicted at left has been reduced. Observe that the greater tuberosity now lies in its normal anatomic position.

mechanical block to abduction and elevation of the arm. Its size bearing no relation to the extent of cuff disruption. In most instances the fracture line involves the floor of the bicipital groove and some changes in the groove subsequent to healing may be anticipated. Such abnormalities comprise spurring, irregularity of the floor and the walls of the groove or even obliteration of portions of the groove.

In the light of the afore mentioned pathology it becomes apparent that anatomic repositioning of the fragments and repair of the torn musculotendinous cuff is essential. This is achieved by exposing the region by a transacromial incision. The fragment is pulled down to its normal site and fixed to the humeral head either by a screw or a wire loop. Repair of the cuff is accomplished by the methods described in Chapter 4.

Fractures of the tuberosity with retraction are strong indications for surgical intervention. Complete anatomic restoration of the bony and soft tissue elements can be achieved only by open operation. Treatment by closed methods with the arm in abduction and external rotation results frequently



FIG 243 Fracture of the greater tuberosity and neck of the humerus. The tuberosity fragment has been displaced upward toward the acromion process by the attached rotator tendons.



FIG. 240 Impacted fracture of the anatomic head often responsible for stiff and painful shoulders. As in depressed fractures of the greater tuberosity this lesion may damage the biceps tendon primarily or (because of changes in the bicipital groove) may interfere with the normal tendon tendon sheath gliding mechanism of the biceps tendon. This patient developed a frozen shoulder.



FIG. 241 Comminuted fracture of the greater tuberosity, with minimal displacement.

In young individuals uncomplicated bicipital tenosynovitis is the most common sequela while in those past middle life the bicipital affection initiates processes which terminate in frozen shoulder.

Treatment of this fracture is similar to that for all impacted fractures. As in depressed fractures of the greater tuberosity the prognosis is poor and undesirable sequelae should be anticipated.

FRACTURES REQUIRING REDUCTION

FRACTURES OF GREATER TUBEROSITY WITH DISPLACEMENT

As a rule, fractures of the greater tuberosity not complicated by dislocation of the humeral head disclose little or no separation (Fig. 241). The same is true of most

greater tuberosity fractures associated with dislocation of the glenohumeral joint. The fragments return to their anatomic position after reduction of the dislocation (Fig. 242). Occasionally after reduction of a dislocation or when no dislocation has occurred the fragments are widely separated from their normal position. They may assume one of two positions: (1) the greater tuberosity may be retracted under the acromion (Fig. 243) or (2) it may be displaced downward and outward.

Fracture with retraction of the tuberosity is equivalent to rupture of the external rotator components of the musculotendinous cuff. There may be massive avulsion of the cuff associated with the lesion. It is impossible to determine the extent of the soft tissue damage without visualization of the subacromial area. The tuberosity retracts under the acromion and acts as a



FIG 246 (*Left*) Abduction fracture of the surgical neck of the humerus. This amount of displacement does not justify manipulation to attain better alignment. (*Right*) Adduction fracture of the surgical neck of the humerus. This amount of displacement is compatible with good function.

pain and muscle spasm preclude adequate examination of the shoulder joint to confirm the diagnosis. Infiltration of the affected area with procaine allows more thorough examination, but abduction of the arm at this time may cause greater separation of the fragments and increase the soft tissue damage. Hence it is contraindicated for diagnostic purposes.

Optimum management is achieved by the open method, utilizing the transacromial route to expose the subacromial region. The fragment is replaced and held in position by wire or screws, and the cuff is repaired by methods previously described. Postoperative treatment then proceeds as in a fracture without displacement.

FRACTURE OF THE LESSER TUBEROSITY WITH RETRACTION

This lesion is exceedingly rare as an isolated lesion, being more frequently encountered in comminuted fractures of the upper end of the humerus and in dislocations of the humeral head. The subscapularis muscle pulls the fragment inward to occupy



FIG 247 Nonimpacted abduction fracture with marked angular deformity. In this case repositioning of fragments is necessary.

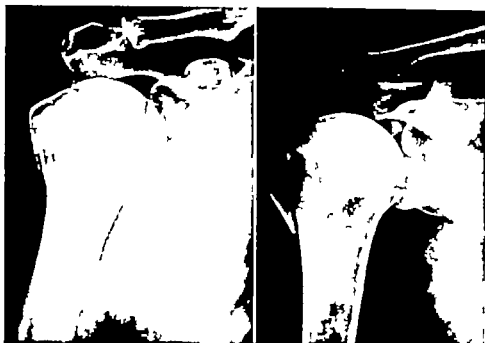


FIG 244 (*Left*) Fracture of neck of the humerus without displacement. Severe comminution of the tuberosities exists. (*Right*) Impacted fracture of the anatomic neck of the humerus with avulsion and slight upward and outward displacement of a portion of the greater tuberosity.



FIG 245 Impacted fracture of the neck of the humerus with extensive comminution of the head.

in dismal failure, leaving the patient with protracted or permanent disability.

Following open reduction of the fracture and repair of the cuff with the arm at the side, the injury is treated in every respect similarly to impacted fractures of the upper end of the humerus. Occasionally the fragment may exhibit extensive comminution so that repositioning and maintenance of reduction as described is not feasible. Excision of the fragments is the procedure of choice in these instances. The rotator cuff is then reattached to the humeral head wherever it will reach with the arm at the side, by methods previously described in Chapter 4. This same approach to the problem may be necessary in old cases in which it is impossible to pull the fragment to its anatomic position. If a dislocation has been present, motion at the shoulder joint is kept below the horizontal for 4 weeks.

In fractures of the greater tuberosity with downward and outward displacement, one must assume that there is also a concomitant rupture of the cuff. As in all acute traumatic lesions about the shoulder joint,



FIG 246 (*Left*) Abduction fracture of the surgical neck of the humerus. This amount of displacement does not justify manipulation to attain better alignment. (*Right*) Adduction fracture of the surgical neck of the humerus. This amount of displacement is compatible with good function.

pain and muscle spasm preclude adequate examination of the shoulder joint to confirm the diagnosis. Infiltration of the affected area with procaine allows more thorough examination, but abduction of the arm at this time may cause greater separation of the fragments and increase the soft tissue damage. Hence, it is contraindicated for diagnostic purposes.

Optimum management is achieved by the open method, utilizing the transacromial route to expose the subacromial region. The fragment is replaced and held in position by wire or screws, and the cuff is repaired by methods previously described. Postoperative treatment then proceeds as in a fracture without displacement.

FRACTURE OF THE LESSER TUBEROSITY WITH RETRACTION

This lesion is exceedingly rare as an isolated lesion being more frequently encountered in comminuted fractures of the upper end of the humerus and in dislocations of the humeral head. The subscapularis muscle pulls the fragment inward to occupy



FIG 247 Nonimpacted abduction fracture with marked angular deformity. In this case, repositioning of fragments is necessary.



FIG 244 (*Left*) Fracture of neck of the humerus without displacement. Severe comminution of the tuberosities exists. (*Right*) Impacted fracture of the anatomic neck of the humerus with avulsion and slight upward and outward displacement of a portion of the greater tuberosity



FIG 245 Impacted fracture of the neck of the humerus with extensive comminution of the head.

in dismal failure leaving the patient with protracted or permanent disability

Following open reduction of the fracture and repair of the cuff with the arm at the side the injury is treated in every respect similarly to impacted fractures of the upper end of the humerus. Occasionally, the fragment may exhibit extensive comminution so that repositioning and maintenance of reduction as described is not feasible. Excision of the fragments is the procedure of choice in these instances. The rotator cuff is then reattached to the humeral head wherever it will reach with the arm at the side by methods previously described in Chapter 4. This same approach to the problem may be necessary in old cases in which it is impossible to pull the fragment to its anatomic position. If a dislocation has been present motion at the shoulder joint is kept below the horizontal for 4 weeks.

In fractures of the greater tuberosity with downward and outward displacement, one must assume that there is also a concomitant rupture of the cuff. As in all acute traumatic lesions about the shoulder joint



FIG 246 (*Left*) Abduction fracture of the surgical neck of the humerus. This amount of displacement does not justify manipulation to attain better alignment. (*Right*) Adduction fracture of the surgical neck of the humerus. This amount of displacement is compatible with good function

pain and muscle spasm preclude adequate examination of the shoulder joint to confirm the diagnosis. Infiltration of the affected area with procaine allows more thorough examination, but abduction of the arm at this time may cause greater separation of the fragments and increase the soft tissue damage. Hence it is contraindicated for diagnostic purposes

Optimum management is achieved by the open method, utilizing the transacromial route to expose the subacromial region. The fragment is replaced and held in position by wire or screws, and the cuff is repaired by methods previously described. Postoperative treatment then proceeds as in a fracture without displacement

FRACTURE OF THE LESSER TUBEROSITY WITH RETRACTION

This lesion is exceedingly rare as an isolated lesion being more frequently encountered in comminuted fractures of the upper end of the humerus and in dislocations of the humeral head. The subscapularis muscle pulls the fragment inward to occupy



FIG 247 Nonimpacted abduction fracture with marked angular deformity. In this case repositioning of fragments is necessary

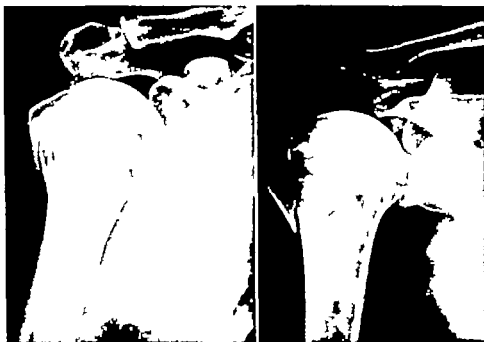


FIG 244 (*Left*) Fracture of neck of the humerus without displacement. Severe comminution of the tuberosities exists. (*Right*) Impacted fracture of the anatomic neck of the humerus with avulsion and slight upward and outward displacement of a portion of the greater tuberosity.



FIG 245 Impacted fracture of the neck of the humerus with extensive comminution of the head.

in dismal failure leaving the patient with protracted or permanent disability.

Following open reduction of the fracture and repair of the cuff with the arm at the side the injury is treated in every respect similarly to impacted fractures of the upper end of the humerus. Occasionally, the fragment may exhibit extensive comminution so that repositioning and maintenance of reduction as described is not feasible. Excision of the fragments is the procedure of choice in these instances. The rotator cuff is then reattached to the humeral head wherever it will reach with the arm at the side, by methods previously described in Chapter 4. This same approach to the problem may be necessary in old cases in which it is impossible to pull the fragment to its anatomic position. If a dislocation has been present motion at the shoulder joint is kept below the horizontal for 4 weeks.

In fractures of the greater tuberosity with downward and outward displacement, one must assume that there is also a concomitant rupture of the cuff. As in all acute traumatic lesions about the shoulder joint,

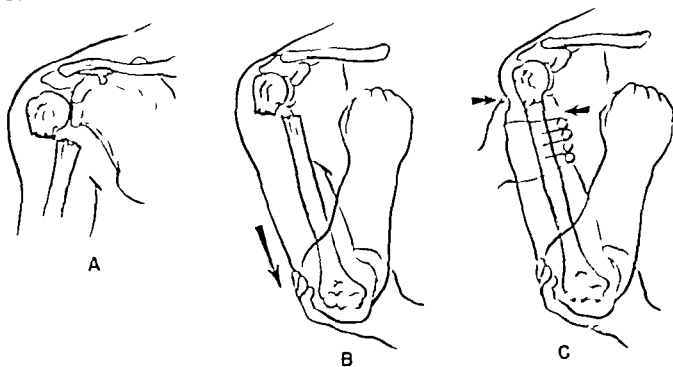


FIG 248 Method of reducing completely displaced fractures of the surgical neck by traction on the slightly flexed and adducted arm

have fallen into proper alignment, traction is released gradually allowing them to engage. The arm is then carefully brought to the patient's side and immobilized by a collar and-cuff sling and swathe (Fig. 248).

Postreduction management is similar to that of unimpacted fractures requiring no reduction. Movement in the fingers, the wrist and the elbow are started immediately. After 2 weeks, sufficient bony healing has taken place to permit antigravity, pendulum exercises. As a rule after 3 weeks all fragments move in unison upon rotating the humeral shaft indicating that movements against gravity may be begun with no fear of displacement.

The great majority of displaced surgical neck fractures can be reduced by the above manipulative method. In most instances, the position of the fragments will not be lost when traction is released and when the arm is brought to the side. In a goodly number, however because of the characteristics of the fragments reduction cannot be maintained without traction.

In such instances the patient is placed in a recumbent position the fracture is reduced as described above and necessary

traction is provided by suspending the flexed and adducted arm from an overhead frame. Skeletal traction (wire or pin through the olecranon) is the simplest and most efficacious form of traction (Fig. 249). It also permits early mobilization of the joints of the fingers and of the wrist. Within 2 weeks healing will have progressed sufficiently to allow all fragments to move in unison upon internal and external rotation of the humeral shaft. This denotes that traction may be removed with safety and the arm may be brought to the side. Subsequent treatment proceeds as in the case of fractures of this region which require no reduction.

In some instances all manipulative measures fail to achieve a satisfactory reduction of the fracture. Traction with the arm in flexion and adduction as described above will not bring about repositioning of the fragment. It becomes necessary in these difficult fractures to resort either to open procedures or to apply traction with the arm in slight abduction. Occasionally, this last method will produce a satisfactory reduction when all other closed methods fail.

The arm is placed in from 45° to 60° ab-

a position between the humeral head and the coracoid process. It now acts as a bony block to internal rotation, flexion and elevation of the arm. Restoration of the fragment to its normal position is essential and can be accomplished only by open operation. An anterior deltoid splitting incision provides adequate exposure for this procedure. As a rule, the fragment is readily tacked in place by interrupted sutures passing through drill holes which have been made in the detached fragment and in the edges of the defect in the humerus.

Postoperative management is the same as in fractures without displacement. Care must be taken to refrain from early forceful external rotation exercises which are likely to pull the lesser tuberosity from its anchorage.

FRACTURES OF THE HUMERAL NECK

Fractures of the humeral neck are common lesions and may occur in all age periods. They are encountered more frequently in adults, only rarely in children. The lesions may be subperiosteal and incomplete without separation of the fragments as is usually observed in children. On the other hand, depending upon the direction the arm takes as the patient falls on the outstretched hand (see Mechanism of Fractures of the Upper End of the Humerus), the humeral shaft may be displaced into an abducted or adducted position in relation to the proximal fragment (humeral head) as shown in Figure 246. Not infrequently the fracture may be a concomitant lesion of extensive comminution of the upper end of the humerus (Fig. 245).

As previously recorded, no impacted fracture, regardless of the type or the degree of deformity, requires reduction. Repositioning of the fragments is warranted only in unimpacted fractures with complete displacement and in those with severe angular disalignment which, if not eliminated or decreased, would provide a mechanical impediment to normal glenohumeral motion (Figs. 246 and 247).

SURGICAL NECK FRACTURES WITH COMPLETE DISPLACEMENT

This is not a common lesion. The proximal fragment may be abducted slightly and rotated externally by the short rotator muscles inserting into the greater tuberosity. As a rule, the head fragment (except for slight abduction) is not materially affected by muscle pull, because the pull of the short external rotators is almost completely counterbalanced by that of the subscapularis muscle.

The distal fragment, completely separated from the head fragment, is displaced upward, forward and inward. Upward displacement is the result of the direction of the initial violence and the pull of long muscles paralleling the shaft of the humerus (deltoid, triceps, biceps and coracobrachialis). Forward displacement is affected by the pectoralis major, the teres major and the latissimus dorsi. Inward displacement by the pectoralis major, the short head of the biceps and the coracobrachialis.

Treatment—Manipulative Reduction with Arm in Adduction and Flexion. Complete muscle relaxation is essential to attain reduction with minimum force and is best attained by general anesthesia. Pentothal Sodium is an excellent agent for this purpose. With the patient in the recumbent position, an assistant makes steady traction on the arm first in line with the long axis of the body. Then traction being maintained, the arm is adducted across the anterior surface of the thorax and flexed in relation to the frontal plane of the body. By this maneuver, the length of the arm is restored and all forces responsible for the deformity are eliminated. Flexion and adduction relax the pectoralis major, the teres major and the latissimus dorsi while steady traction overcomes the deforming forces of the long muscles paralleling the humerus.

After arm length has been restored, the operator places his hand in the axilla and pushes the upper end of the shaft fragment outward while firm inward pressure is made on the head fragment. When the fragments

rovascular structures in the axilla are apt to be severely stretched and traumatized. Moreover, abduction maneuvers tend to separate the fragments and to increase the forces responsible for displacement of the distal fragment. Also the pectoralis major, the teres major and the latissimus dorsi are stretched unduly. Only in exceptional cases which fail to respond to reduction methods is mild abduction of the extremity (from 45° to 60°) permissible to effect a reduction. However, the reduction always must be accompanied by continuous traction.

Abduction apparatus, designed with or without traction in order to make the patient ambulatory, never should be used. They are cumbersome and difficult to apply; moreover, they fail to maintain proper reduction. In fact, they increase the deformity and are responsible for marked functional disability. The author has seen one patient who was treated by the abduction or airplane splint, healing took place with pronounced abduction deformity of the distal fragment. When the arm is lowered, the upper end of the fragment abuts against the inferior glenoid rim so that the arm cannot be brought to the side but projects from the trunk at an angle of 45° .

FRACTURES OF THE ANATOMIC NECK OF THE HUMERUS

Fractures through the anatomic neck, as single lesions, are rare and usually occur in combination with other fractures of the upper end of the humerus and with fracture dislocation of the humeral head. In many instances the capital fragment includes one or both tuberosities. It usually occurs in adults past middle life, rarely in children. Depending upon the direction which the extremity takes as the patient falls on the outstretched hand, the capital fragment is forced into an abducted or adducted position, the abduction type being slightly more common. Generally, the fragments are impacted since the fracturing force drives the upper end of the humeral shaft into the cancellous bone of the capital fragment.

The intracapsular fragment in spite of the noted impaction may be severed from all its blood supply. Fortunately, in many instances it maintains continuity with some of the other fragments, particularly the lesser tubercle which may have adequate circulation. Hence, some nourishment to the intracapsular fragment is assured. Diminished or complete obliteration of the blood supply to this fragment may be followed by avascular necrosis, which may not manifest itself for from 18 to 24 months after the injury. This complication results in a stiff and painful shoulder joint. In the light of the above information, one must not consider these fractures lightly. The prognosis should be guarded, and the disabling complication of avascular necrosis of the anatomic head should be anticipated in a certain percentage of these cases.

Treatment. Regardless of the type of fracture or the extent of the deformity, no reduction is indicated if the fragments are impacted. Such cases are treated similarly to other impacted fractures of the upper end of the humerus. Repositioning of the fragments is justified in unimpacted fractures with gross angular deformities or with complete displacement.

Abduction fracture of the anatomic neck with no impaction and complete displacement or gross angulation are reduced by traction on the adducted and flexed arm and manipulation as described for fractures of the surgical neck. Postreduction treatment is the same as for fractures in this region requiring no reduction. If reduction cannot be maintained without continuous traction, skeletal traction is employed, as described in surgical neck fractures.

At no time must one be tempted to treat these lesions on an abduction frame or splint. These apparatus increase the deformity and allow the fragments to heal with gross malalignment. Occasionally, when closed methods fail to produce the desired reduction, operative intervention is justifiable.

Adduction fractures with gross angular



FIG. 249 Method of continuous traction to maintain position of fragments following reduction by manipulation as depicted in Figure 248

duction (60° must not be exceeded), and the forearm is flexed at the elbow to 90° . Traction is made along the shaft of the humerus by means of a pin or a wire through the olecranon. Sufficient traction is applied at the fracture site to restore alignment. Distraction must be avoided (Fig. 250). In this position the extremity is in balance as regards the various forces producing the deformity. From 45° to 60° of abduction does not put the pectoralis major under undue tension whereas traction restores length and overcomes the forces of the long muscles of the arm. After 2 or 3 weeks, healing advances sufficiently to assure maintenance of reduction without traction. All apparatus is then removed; the patient becomes ambulatory and the fracture is treated in the same way as fractures requiring no reduction.

DISCUSSION OF ABDUCTION TREATMENT
Manipulative measures with the arm in wide abduction in surgical neck fractures are dangerous procedures, because the neu-

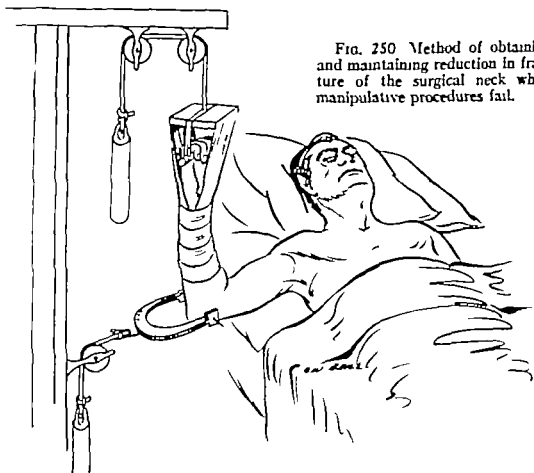


FIG. 250 Method of obtaining and maintaining reduction in fracture of the surgical neck when manipulative procedures fail.

The shaft may be adducted and impacted into the inner portion of the epiphysis, or the epiphyseal fragment may include a small triangular portion of the metaphysis or even a large segment of the upper end of the diaphysis (Fig 251)

Disturbances of growth may occur following trauma to the epiphyseal plate. The extent of epiphyseal damage and subsequent growth disturbance cannot be assayed properly by the degree of separation discernible on radiographic examination. Gross displacement may be followed by little or no disturbances of bone growth, while marked growth disturbance and gross deformity may occur subsequent to minor displacements. It is evident that treatment of these lesions demands great gentleness. Further damage to the epiphyseal plate must be avoided.

TREATMENT

Management of these lesions should be guided by the knowledge that nature tends to adjust and minimize all bone deformities by subsequent bone growth and that all manipulative measures regardless of how gently they are executed, inflict further injury to the epiphyseal plate. Reduction, if

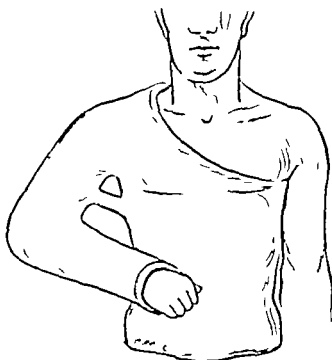


FIG 252 Plaster-of-Paris shoulder spica used to immobilize the extremity following reduction of epiphyseal fracture.

necessary is accomplished more easily in early than late lesions and less force effects a reduction in early than in late epiphyseal separation.

Lesions with slight displacement of the



FIG 253 (Left) Complete epiphyseal separation. Several attempts at reduction by manipulative measures failed. (Right) Repositioned by surgical intervention of the epiphysis shown at left. Internal fixation was necessary because stability of the fragments could not be achieved otherwise.

deformity or complete displacement are reduced with the patient in a recumbent position under a general anesthesia. Shortening of the limb is overcome by traction, with the arm slightly flexed and abducted. Length

is gained by traction. The fracture line usually begins at the base of the greater tuberosity and continues upward and inward to the middle of the epiphysis, then follows the line of the anatomic neck.

The most common injury to the upper end



FIG. 251 (*Left*) Epiphyseal fracture of the humerus with typical adduction deformity. The epiphyseal fragment includes a large triangular portion of the upper end of the diaphysis. (*Right*) Following replacement of the epiphysis shown at left.

having been restored the fragments are manipulated into satisfactory alignment. By releasing the traction the fragments are permitted to engage. Postreduction treatment is the same as that of fractures of this region which requires no reduction. Anatomic neck fractures occurring in conjunction with dislocation of the glenohumeral joint are discussed under fracture-dislocations.

SEPARATION OF THE HUMERAL EPIPHYSIS

Three centers of ossification—one for the head, the greater tuberosity and the lesser tuberosity—coalesce at the sixth or the seventh year to form the upper end of the humerus or upper humeral epiphysis which fuses with the shaft about the twentieth year. The epiphyseal plate is roughly con-

cave of the humerus in the second decade of life is separation of the upper humeral epiphysis. The mechanism of production is similar to that of fractures of the surgical neck. In the vast majority of cases the shaft is adducted in relation to the head. Occasionally, however, an abduction type of displacement is encountered.

There may be only minimal separation of the epiphysis with little or no displacement demonstrable in the radiographic examination by widening of the epiphyseal space in the region of the base of the greater tuberosity and a slight inward tilt of the capital mass. Complete separation may occur with displacement of the diaphysis inward upward and forward. This lesion resembles in every respect adduction fracture through the anatomic neck with complete displacement. Incomplete displacement may occur



FIG 255 (Left) Fracture-dislocation of the humerus. No displacement of the greater tuberosity fragment has occurred there also exists a fracture through the anatomic neck. (Center) Fracture-dislocation of the humerus with the greater tuberosity in approximately its normal position relative to the scapula. However the head had traveled a considerable distance from the glenoid cavity. Closed methods to reduce this dislocation failed. Open measures revealed the biceps tendon displaced posteriorly, obstructing replacement of the head. A vertical tear of the musculotendinous cuff was found in the interval between the subscapularis and supraspinatus muscles. (Right) Fracture depicted (center) reduced greater tuberosity fixed to the head with 1 screw. The cuff was repaired.

duction in such instances is warranted. In late cases, however, when great force must be employed to mobilize the fragments for repositioning, one is justified in accepting the deformity in lieu of traumatizing the epiphyseal plate and assuring subsequent bone-growth disturbance.

Complete displacement of the upper humeral epiphysis demands reduction. Every attempt should be made to reduce the lesion as soon as possible after the injury. Complete muscle relaxation is best attained with a general anesthesia.

With the patient in the recumbent position an assistant makes slow steady traction on the abducted (from 45° to 60°) and flexed arm. Moderate traction is maintained until shortening of the arm is overcome. The operator then places both hands in the axilla so that the fingers are on the upper and inner end of the diaphysis and the thumbs are on the outer aspect of the capital fragment. He then forces the shaft

outward and backward. Traction is then released to allow engagements of the fragments. Reduction is maintained best by a plaster jacket holding the arm in from 30° to 60° abduction, 30° forward flexion mid way between internal and external rotation (Fig 252).

After 3 or 4 weeks the jacket is removed and the arm is brought to the side and suspended in a collar-cuff sling. At the end of 2 weeks free use of the extremity is permissible. As a rule, special exercises and other physical measures are not essential in the postreduction management of children.

Occasionally it is impossible to reduce the displacement or to maintain it without continuous traction. In such instances traction with the patient in bed should be employed as described for fractures of the surgical neck.

Open reduction is justified when it is impossible to effect a reduction by manipula-

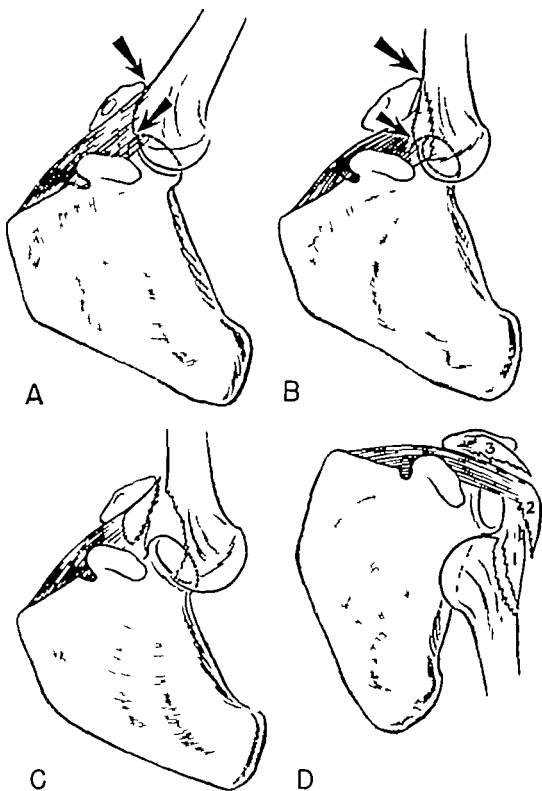


FIG 254 Mechanism of dislocation associated with fracture of the greater tuberosity. The different positions in which the greater tuberosity may be found: (1) the tuberosity follows the head; (2) it retains its normal relation to the scapula; (3) it has been retracted under the acromion.

humeral epiphysis should not be disturbed. Fixation of the extremity with a collar-cuff sling and a swathe about the body affords adequate immobilization. The amount of displacement does not tend to increase. Thus, more rigid immobilization is not

necessary. There is sufficient healing to permit free use of the extremity after 3 weeks.

If seen early, most lesions with moderate displacement can be reduced by gentle traction and manipulative maneuvers and re-



FIG 257 (*Left*) Fracture-dislocation with impaction of the head fragment. Manipulative methods to effect reduction may disengage the fragments, thereby adding to the difficulty of reduction (*Right*) Fracture-dislocation with marked displacement of the head fragment. All nourishment to the head is severed if the head is replaced on the end of the shaft, avascular necrosis is a certainty. The greater tuberosity is pulled under the acromion by the external rotators

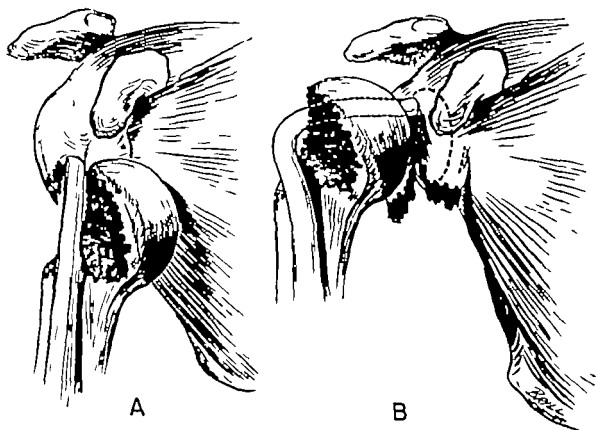


FIG 258 Schematic drawing illustrating fracture-dislocation in which the tuberosities have been pulled off the humerus and, with the cuff intact have dropped in front of glenoid cavity (A)—thereby obstructing reduction (B) The biceps tendon usually is displaced posteriorly



FIG 256 Fracture-dislocation of the humerus in which the greater tuberosity is completely displaced from its normal bony position but has followed the shaft of the humerus. In this instance the cuff may be severely stretched across the glenoid cavity or it may be ruptured.

tion or traction or when healing has progressed to the point that it is impossible to mobilize the fragments without serious damage to the epiphysis and the soft tissue structures. The surgeon should be cognizant of the fact that open reduction is followed invariably by disturbance of bone growth and he should realize further that this sequela will be of less significance to the patient than the deformity of the displaced unreduced epiphysis (Fig 253).

FRACTURE-DISLOCATIONS OF THE HUMERUS

Fractures of the upper end of the humerus occurring in conjunction with dislocation of the glenohumeral joint constitute the most difficult of all shoulder injuries. In general two types of fractures may occur

with dislocation: (1) fracture of the greater tuberosity and (2) fracture of the humeral head or neck.

FRACTURE OF THE GREATER TUBEROSITY

This lesion is the most common complication and occurs in from 25 to 30 per cent of all dislocations of the glenohumeral joint. Its mechanism of production denotes that it must be accompanied necessarily by extensive damage of the musculotendinous cuff (Fig 254). A rough evaluation of the soft tissue injury may be ascertained by the radiographic characteristics of the fracture.

In most cases the tuberosity fragment lies in its normal anatomic position in relation to the acromion and retains its attachment to the musculotendinous cuff. A tear paralleling the fibers of the cuff in the interval between the supraspinatus and the subscapularis tendons constitutes the usual soft tissue injury. The humeral head is dislocated but may be within the capsule and corresponds to Codman's "false-dislocation." Reduction of the dislocation by the methods described affects anatomic repositioning of the fragments. Treatment is the same as if no fracture existed. Abduction splints and other appliances are not necessary and are harmful.

Occasionally with the tuberosity in approximately its normal position and within the musculotendinous cuff the head may occupy a position at a great distance from its normal berth. Such displacement may tear the biceps tendon from its insertion into the superior glenoid rim or displace it to a position posterior to the head. In this abnormal position the tendon may act as a mechanical impediment to reduction and require operative intervention to achieve replacement of the humeral head (Fig 255).

Retraction of the fracture fragment under the acromion associated with dislocation is indicative of extensive tearing of the musculotendinous cuff. Management of this lesion entails repositioning of the fragments

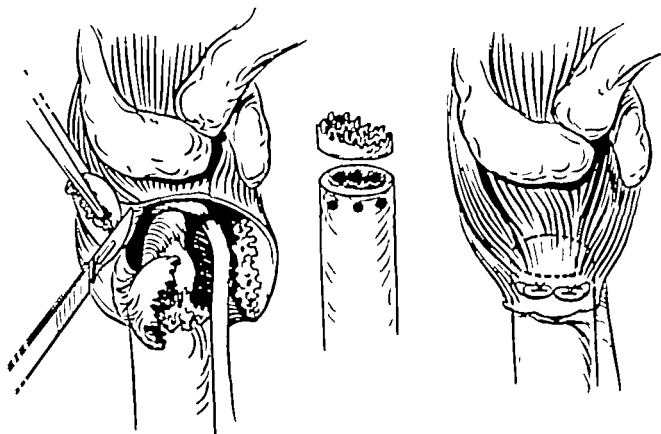


FIG. 261 Operation to restore function of the rotator muscles and stability of the glenohumeral joint. The head and tuberosity fragments are removed the musculotendinous cuff and inferior capsule are sutured through drill holes made in the shaft to a lower level on the shaft of the humerus.

comprises pronounced disorganization of the osseous and soft tissue elements of the joint the jagged ends of the fragments may injure the brachial plexus and vessels in the axilla Injury to these structures may occur at the time of the initial violence or during the course of manipulative maneuvers Some workers advocate closed methods to effect reduction chiefly by application of strong traction to the abducted arm and manipulation of the head into the glenoid cavity (Figs 257-260)

It becomes evident that such procedures further jeopardize the neurovascular elements and are likely to inflict more trauma to the musculotendinous cuff Moreover any existing impaction of the fragments may be broken up thereby adding greater difficulty to the problem of reduction Above all methods such as the Kocher or the Hippocratic which utilize the humerus as a lever should be condemned

Visualization of the pathology is the most logical approach to this difficult problem. McLaughlin's transacromial incision provides adequate exposure of the subacromial area and ample room for whatever operative work is deemed necessary

Repositioning of the fragments into normal alignment and replacement of the head in the glenoid cavity should be done with the utmost gentleness It may be necessary to divide the insertion of the pectoralis major and the subscapularis muscles to mobilize the fragments adequately Every effort should be made to preserve soft tissue and periosteal attachments of the head fragments Pieces of bone completely devoid of soft tissue should be removed The fragments after being assembled are fixed to one another by wire or stout cat gut sutures passing through drill holes in the adjacent fragments Nicola's procedure may be utilized to advantage in stabilizing the ana



FIG 259 Fracture-dislocation with wide separation of fragments. Head fragment and tuberosities were removed and the cuff was anchored to the end of the shaft of the humerus.

and repair of the disrupted cuff which can be achieved only by open methods. These have been described in the discussion on fractures of the greater tuberosity with retraction.

In some instances the fractured tuberosity follows the humeral head at some distance from its anatomic position in relation to the scapula (Fig. 256). In such cases, one can assume that the external rotators are severely stretched over the glenoid cavity or torn from the tuberosity. Following reduction, the operator must be on the alert for clinical evidence of a torn cuff. If such evidence is apparent, exploration of the subacromial area through a transacromial incision is justified and the torn cuff is repaired by the method already described.

FRACTURES OF THE HEAD AND THE NECK OF THE HUMERUS WITH DISLOCATION

These lesions usually occur in individuals past middle life and constitute the most serious of all complications of dislocation of the glenohumeral joint. The pathology

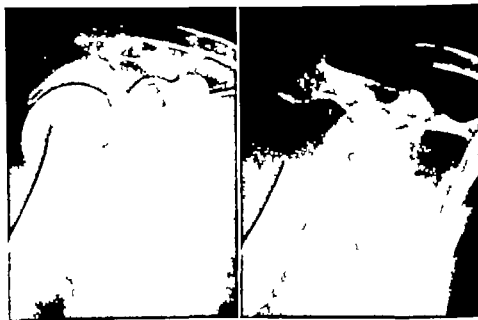


FIG 260 (*Left*) Fracture-dislocation with complete detachment and wide separation of fragments. The head of humerus and the tuberosities were removed. The cuff and the inferior capsule were reattached to head of shaft of humerus. (*Right*) Following removal of head and tuberosities and reattachment of cuff and capsule to end of shaft of patient shown at left.

and performed in the plint which is worn continuously for 4 weeks and at night for 2 additional weeks

The results of this procedure have proven

to be highly satisfactory in the hands of some workers. However the author has found McLaughlin's operation to be more satisfactory

BIBLIOGRAPHY

- Atken A. I. End results fractures of proximal humeral epiphysis J Bone & Joint Surg 18 1036 1936
- Codman E. A. The Shoulder Boston Thomas Todd, 1934
- Curtis F. E. and Branch, H. E. Extra articular arthrodesis of shoulder J Bone & Joint Surg 19 511 1937
- Fischer W. R. Fracture of scapula requiring open reduction report of a case J Bone & Joint Surg. 21 459 1939
- Frankau Manipulative reduction fractures of the surgical neck of the humerus Lancet 2 755 1933
- Harmon P. H. and Baker D. R. Fracture of scapula with displacement J Bone & Joint Surg 25 834 1943
- Howard N. J. and Eloesser L. Treatment of fractures of the upper end of the humerus J Bone & Joint Surg 16 1 1934
- Jones L. Observations on the anatomy and physiology with analysis of reconstruction operation following extensive injury Surg., Gynec. & Obst 75 433 1942
- McLaughlin H. L. Treatment of shoulder injuries regional orthopedic surgery and fundamental orthopedic problems Ann Harbor Mich. Edwards 1947
- Magnuson P. B. Fractures ed. 5 Philadelphia Lippincott 1949
- Michaels L. S. Comminuted fracture dislocation of the shoulder J Bone & Joint Surg 26 363 1944
- Murray G. A method of fixation for fracture of the clavicle J Bone & Joint Surg 22 616 1940
- Watson Jones R. Fractures and Joint Injuries, Baltimore Williams & Wilkins 1946
- Watson Jones R. Extra-articular arthrodesis of shoulder J Bone & Joint Surg. 15 862 1933
- Wilson, P. D. and Cochrane W. A. Fractures and Dislocations Philadelphia Lippincott 1925

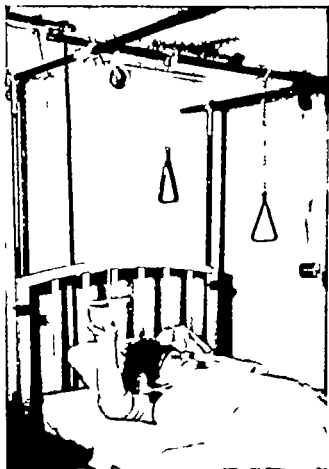


FIG 262 Patient of Figure 260 following operation with arm in balanced suspension allowing early active motion and development of tone and control in muscles of the shoulder girdle

tomic head on the shaft. Repair of the cuff tears usually ensures maintenance of reduction of the fragments.

Not infrequently the anatomic head is found lying free in the soft tissues completely severed from any source of nourishment or comminution is so great that avascular necrosis is a certainty. In such instances removal of the head fragment is unavoidable despite the realization that the procedure causes great functional disability. Without the humeral head the short rotators are no longer able to stabilize the humerus on the glenoid cavity making elevation of the arm impossible.

Several ingenious procedures have been designed to overcome this mechanical disadvantage. McLaughlin advocates transplantation of the insertion of the musculo-

tendinous cuff to a lower level on the humeral shaft and reconstruction of the disrupted cuff so that, with the inferior portions of the fibrous capsule, it surrounds the upper end of the humerus completely. He further recommends removal of the outer half of the acromion and suturing the central tendon more mesially in order to increase the leverage power of this muscle for the elevation of the extremity. The author has performed this operation many times and found it to be most satisfactory (Fig 261).

Postoperative management should aim at mobilization of the extremity as soon as sufficient soft tissue healing permits. The arm is placed in balanced suspension (Fig 262), and motion is started immediately. After from 10 to 12 days the arm is brought to the side and stooping exercises are begun. A rigid regimen of progressive exercises must be enforced to develop the muscle tone and control necessary to stabilize the new glenohumeral articulation.

Lawrence Jones has devised a procedure similar in principle to the one just described. The subacromial area is exposed by a Cubbins incision (see *Operative Approaches to the Shoulder Joint*) and the musculotendinous cuff is freed from bony attachments. All fragments of the upper end of the humerus are removed and the end of the shaft is rounded. Two flaps are created from the musculotendinous cuff: an anterior flap comprising the subscapularis muscle, and the posterior flap consisting of the supraspinatus, the infraspinatus and the teres minor muscles. Approximately 2 inches below the rounded end of the humerus two vertical grooves are made in the humeral shaft through the entire thickness of the cortical bone. The flaps are sutured in their respective grooves and to one another by a strip of fascia lata previously obtained from the thigh.

Postoperative treatment entails fixation of the extremity in 135° abduction by means of an abduction splint. Exercises are begun as soon as soft tissue healing is complete.

and performed in the plint which is worn continuously for 4 weeks and at night for 2 additional weeks

The results of this procedure have proven

to be highly satisfactory in the hands of some workers. However, the author has found McLaughlin's operation to be more satisfactory

BIBLIOGRAPHY

- Atken A. I. End results fractures of proximal humeral epiphysis *J Bone & Joint Surg.* 18 1036 1936
- Codman E. A. *The Shoulder* Boston Thomas Todd, 1934
- Curtis F. E. and Branch, H. E. Extra articular arthrodesis of shoulder *J Bone & Joint Surg.* 19 511 1937
- Fischer W. R. Fracture of scapula requiring open reduction report of a case *J Bone & Joint Surg.* 21 459 1939
- Frankau Manipulative reduction fractures of the surgical neck of the humerus *Lancet* 2 755 1933
- Harmon P. H. and Baker D. R. Fracture of scapula with displacement *J Bone & Joint Surg.* 25 834 1943
- Howard, N. J., and Eloesser L. Treatment of fractures of the upper end of the humerus *J Bone & Joint Surg.* 16 1 1934
- Jones L. Observations on the anatomy and physiology with analysis of reconstruction operation following extensive injury *Surg Gynec & Obst.* 75 433 1942
- McLaughlin H. L. Treatment of shoulder injuries regional orthopedic surgery and fundamental orthopedic problems Ann Harbor Mich. Edwards 1947
- Magnuson P. B. *Fractures* ed. 5 Philadelphia Lippincott 1949
- Michaelis L. S. Comminuted fracture dislocation of the shoulder *J Bone & Joint Surg.* 26 363 1944
- Murray G. A method of fixation for fracture of the clavicle *J Bone & Joint Surg.* 22 616 1940
- Watson Jones R. *Fractures and Joint Injuries* Baltimore Williams & Wilkins 1946
- Watson Jones R. Extra articular arthrodesis of shoulder *J Bone & Joint Surg.* 15 862 1933
- Wilson P. D., and Cochrane W. A. *Fractures and Dislocations* Philadelphia, Lippincott 1925

Shoulder Pain of Neurogenic Origin and Obstetric Paralysis

REFLECTED PAIN

ANATOMIC CONSIDERATIONS

CERVICAL SEGMENT OF THE SPINAL COLUMN
BRACHIAL PLEXUS

TOPOGRAPHIC AND VARIATIONAL ANATOMY OF THE SUPRACLAVICULAR REGION

SYRINGOMYELIA

HYPERTROPHIC CERVICAL PACHYMENINGI
TIS

PLATYBASIA (BASILAR COMPRESSION)

HERPES ZOSTER

INFLAMMATORY LESIONS

GUILLAIN BARRÉ SYNDROME

LESIONS ASSOCIATED WITH PATHOLOGIC DIS- ORDERS OF THE VERTEBRAL COLUMN

TUBERCULOSIS

RHEUMATOID ARTHRITIS

LESIONS ASSOCIATED (*Continued*)

HYPERTROPHIC ARTHRITIS

PRIMARY AND METASTATIC NEOPLASMS

TRAUMATIC LESIONS

PROTRUSION OF THE INTERVERTEBRAL DISK
LESIONS SITUATED OUTSIDE THE VERTEBRAL
COLUMN

CERVICAL RIB SYNDROME

SCALenus ANTICUS SYNDROME

SUBCORACOID PECTORALIS MINOR SYNDROME
AND COSTOCLAVICULAR SYNDROME

TUMORS

INFLAMMATORY LESIONS

SERUM NEURITIS

TRAUMATIC LESIONS

OBSTETRICAL PARALYSIS

INTRODUCTION

Pain projected to the shoulder and the arm may be produced by a host of etiologic factors. Failure to apprehend the cause and the mechanism of the pain has favored the adoption of such vague nonspecific terms as brachial neuritis and brachialgia. Nachlas however emphasizes that brachialgia is a symptom complex characterized by pain radiating to the shoulder and the arm and in no way denotes a causative agent.

This observer also points out that the syndrome may result from irritation of sensory nerve fibers from the sensorium to the peripheral point of reference. Accumulated knowledge of this problem and more adequate comprehension of the mechanisms of the heterogeneous causative factors re-

veal that in the vast majority of instances the syndrome is produced by compression of the spinal cord (spinal cord tumors) or nerve roots (herniated intervertebral disks), lesions which are amenable to treatment usually followed by gratifying results.

Clinical and experimental investigations disclose that segmental reference of pain to the shoulder region and the arm does not always result from sensory nerve involvement of the central or peripheral nervous system. It may be produced by pathologic processes affecting such tissues of mesodermal origins as ligaments, periosteum, fascia, tendons and muscles. Moreover it is generally known that disorders of the heart, lungs, great vessels, pleura, diaphragm and even abdominal viscera may give rise to the same syndrome.

Thus, the numerous causative factors can be grouped into two categories (1) those of neurogenic origin and (2) those of non neurogenic origin.

Lesions of neurogenic origin are characterized by such objective manifestations of central and peripheral nerve involvement as wasting and motor weakness of muscles, abnormal reflex reactions, fibrillations, sensory alterations adhering more or less to a segmental pattern and finally distinctive features of pain which will be considered subsequently.

REFERRED PAIN

Lewis and Kellgren were able to produce pain identical to that of angina pectoris by injecting the left eighth cervical or the first dorsal interspinous ligament with 5 per cent saline solution. The induced pain produced

the same segmental pattern as angina pectoris. In some instances numbness and tingling of the hands as well as hyperalgesia of the skin along the inner aspect of the arm were also concomitant features. The investigators have demonstrated that muscular rigidity and deep tenderness in addition to segmental pain can be produced by stimulating somatic structures and viscera. Eaton points out however that hyperalgesia and paresthesias evoked by somatic and visceral stimulation usually indicate pathologic disorders of the central or peripheral nervous system.

Kellgren noted that by injecting a specific muscle he provoked pain in a definite area, following a segmental pattern. The pain distribution in a given segment was generally constant, unless it was of unusual severity, then it spread to other segments.

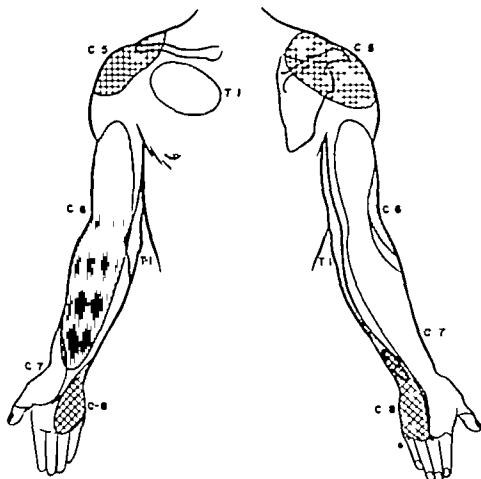


FIG 263 Distribution of pain following injection of rhomboids (horizontal square hatching), flexor carpi radialis (vertical hatching), abductor pollicis longus (light stippling), third dorsal interosseous (oblique square hatching) and first intercostal muscle (dark stippling) (Kellgren Clin. Sc. 3 183 Redrawn with modifications.)

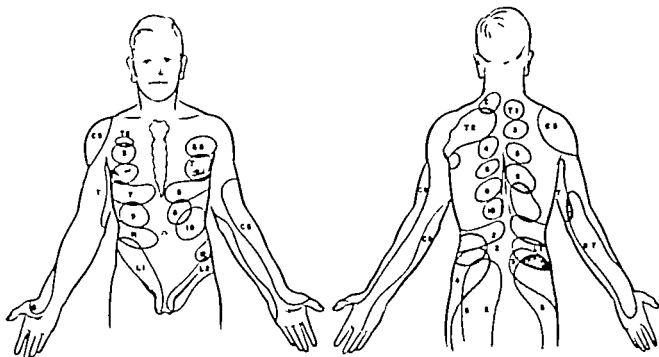


FIG 264 Segmental areas of deep pain developed by the injection of the corresponding interspinous ligaments (diagrams constructed from Kellgren) (Lewis Pain Macmillan p 123 Redrawn with modifications.)

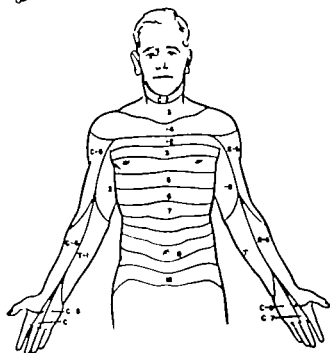


FIG 265 Dermatomes in the trunk and upper extremities. Anterior aspect (Haymaker W and Woodhall B Peripheral Nerve Injuries Philadelphia Saunders p 21)

From these observations Kellgren concluded that the segmental pattern of referred pain following stimulation of a muscle is determined by the nerve root or roots supplying the muscle. The pattern will include other muscles supplied by the same nerve root or roots.

For example, the ninth intercostal nerve

supplies the multiridus, the rectus and the intercostal muscles. By injecting any one of these muscles with hypertonic saline solution, Kellgren could evoke pain in all three areas. Moreover by injecting the interspinous ligaments of the vertebral column at each segmental level, he induced pain in the extremities and the trunk and the pain followed a specific segmental pattern no different from that obtained by stimulating the individual muscles supplied by the same spinal segments (Figs 263 and 264).

Deep segmental pain according to the above investigators, is independent of the superficial or cutaneous form described by Foerster and by Head. Their segmental pattern is similar but discloses some differences particularly in the extremities. Segmental sensory areas of the superficial system were designated dermatomes by Head and skin areas by Sherrington. Dermatomes extend to a lower level in the extremities than the pattern of deep segmental pain (compare Figures 265, 266 and 267 with Figure 264).

Inman and Saunders have made some significant observations referable to deep pain when mesodermal tissues were stimulated by chemical or mechanical agents. Deep radiating pain was produced by stimulating periosteum, fascia, bones, ligaments and tendons. This pain was segmental in nature and unrelated to the peripheral sensory nerves or their segmental cutaneous distribution. The investigators postulate that this type of deep pain may originate in fine sensory nerve endings located in the deep structures and that local axon reflex or misinterpretation by the central nervous system may be responsible for the radiation. Inman and Saunders contend that irritation of deep mesodermal tissues produces deep radiating pain whose pathways correspond to the segmental innervation of the periosteum, ligaments and attachments of muscles; also that these pathways are continuous and extend the entire length of the limb. They designated these deep segmental areas as "sclerotomes."

Dermatomal patterns of cutaneous distribution differ from those of sclerotomes. The former are located on the preaxial and the postaxial borders of the extremity, whereas as previously noted the latter extend distally for almost the entire length of the limb. However a dermatome may overlie a limited segment of the corresponding sclerotome. Pain produced by irritation of deep mesodermal tissue is deep, dull and boring in nature; there is no evidence of cutaneous sensory disturbances, muscular weakness or atrophy or abnormal reflex phenomena.

The experiments of both Lewis and Kell

gren and Inman and Saunders reveal that irritation of the mesodermal structure is accompanied by muscle soreness and rigidity and tenderness over ligaments and bony prominences in the pathways of deep radiation. It is apparent that tenderness may be

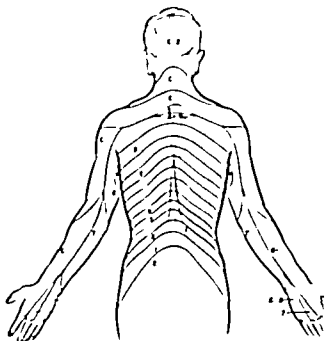


FIG. 266 Dermatomes in the trunk and upper extremities. Posterior aspect. (Haymaker and Woodhall *Peripheral Nerve Injuries*, Philadelphia, Saunders p. 19.)

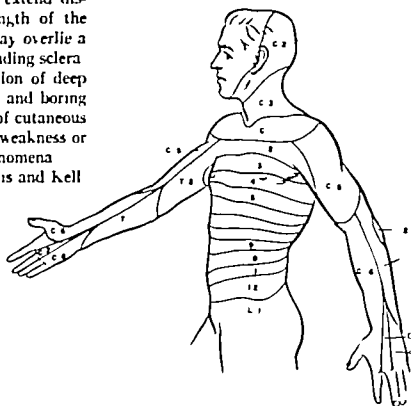


FIG. 267 Dermatomes in the trunk and upper extremities. Lateral aspect. (Haymaker and Woodhall *Peripheral Nerve Injuries*, Philadelphia, Saunders p. 20.)

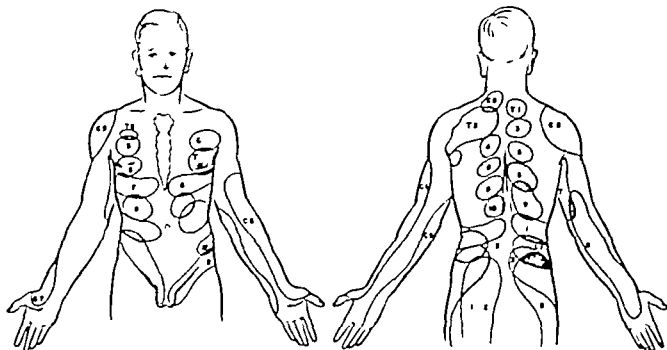


FIG 264 Segmental areas of deep pain developed by the injection of the corresponding interspinous ligaments (diagrams constructed from Kellgren) (Lewis Pain, Macmillan p 123 Redrawn with modifications.)

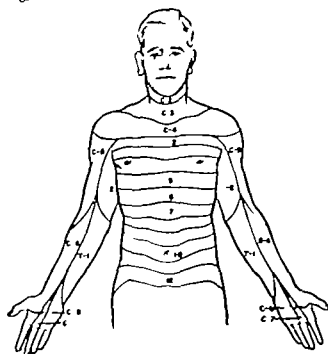


FIG 265 Dermatomes in the trunk and upper extremities. Anterior aspect. (Haymaker W and Woodhall B Peripheral Nerve Injuries Philadelphia Saunders p 21)

From these observations Kellgren concluded that the segmental pattern of referred pain following stimulation of a muscle is determined by the nerve root or roots supplying the muscle. The pattern will include other muscles supplied by the same nerve root or roots.

For example the ninth intercostal nerve

supplies the multijidus the rectus and the intercostal muscles. By injecting any one of these muscles with hypertonic saline solution Kellgren could evoke pain in all three areas. Moreover by injecting the interspinous ligaments of the vertebral column at each segmental level he induced pain in the extremities and the trunk and the pain followed a specific segmental pattern no different from that obtained by stimulating the individual muscles supplied by the same spinal segments (Figs 263 and 264).

Deep segmental pain according to the above investigators, is independent of the superficial or cutaneous form described by Foerster and by Head. Their segmental pattern is similar but discloses some differences particularly in the extremities. Segmental sensory areas of the superficial system were designated dermatomes by Head and skin areas by Sherrington. Dermatomes extend to a lower level in the extremities than the pattern of deep segmental pain (compare Figures 265, 266 and 267 with Figure 264).

Inman and Saunders have made some significant observations referable to deep pain when mesodermal tissues were stimulated by chemical or mechanical agents. Deep radiating pain was produced by stimulating periosteum, fascia, bones, ligaments and tendons. This pain was segmental in nature and unrelated to the peripheral sensory nerves or their segmental cutaneous distribution. The investigators postulate that this type of deep pain may originate in fine sensory nerve endings located in the deep structures and that local axon reflex or misinterpretation by the central nervous system may be responsible for the radiation. Inman and Saunders contend that irritation of deep mesodermal tissues produces deep radiating pain whose pathways correspond to the segmental innervation of the periosteum, ligaments and attachments of muscles, also that these pathways are continuous and extend the entire length of the limb. They designated these deep segmental areas as "sclerotomes."

Dermatomal patterns of cutaneous distribution differ from those of sclerotomes. The former are located on the preaxial and the postaxial borders of the extremity, whereas as previously noted the latter extend distally for almost the entire length of the limb. However, a dermatome may overlie a limited segment of the corresponding sclerotome. Pain produced by irritation of deep mesodermal tissue is deep, dull and boring in nature; there is no evidence of cutaneous sensory disturbances, muscular weakness or atrophy or abnormal reflex phenomena.

The experiments of both Lewis and Hell

gren and Inman and Saunders reveal that irritation of the osseal structures is accompanied by muscle soreness and rigidity and tenderness over ligaments and bony prominences in the pathways of deep radiation. It is apparent that tenderness may be

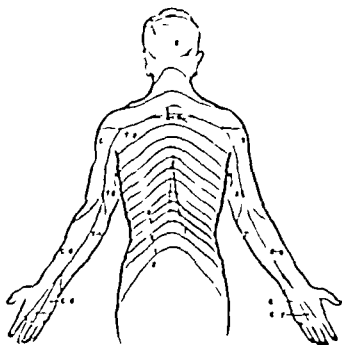


FIG. 266 Dermatomes in the trunk and upper extremities. Posterior aspect. (Haymaker and Woodhall, *Peripheral Nerve Injuries*, Philadelphia, Saunders, p. 19.)

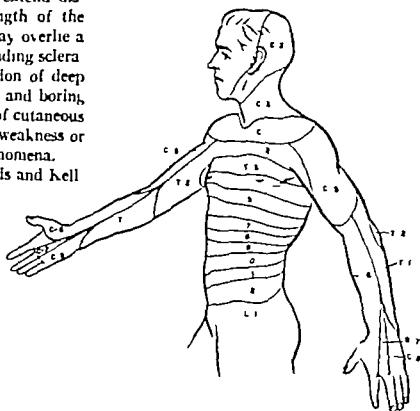


FIG. 267 Dermatomes in the trunk and upper extremities. Lateral aspect. (Haymaker and Woodhall, *Peripheral Nerve Injuries*, Philadelphia, Saunders, p. 20.)

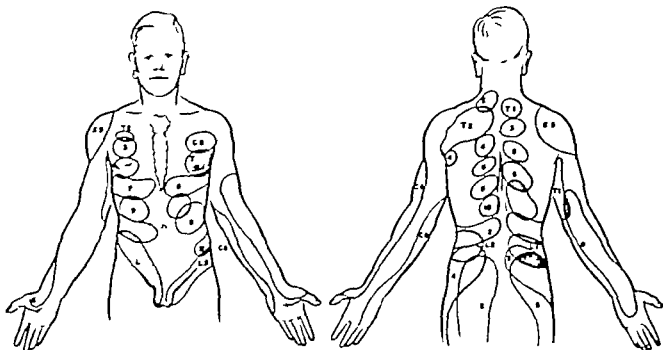


FIG. 264 Segmental areas of deep pain developed by the injection of the corresponding interspinous ligaments (diagrams constructed from Kellgren) (Lewis Pain Macmillan p. 123 Redrawn with modifications.)

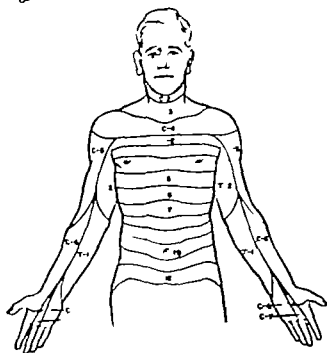


FIG. 265 Dermatomes in the trunk and upper extremities. Anterior aspect. (Haymaker W., and Woodhall B. *Peripheral Nerve Injuries* Philadelphia. Saunders, p. 21)

supplies the multifidus the rectus and the intercostal muscles. By injecting any one of these muscles with hypertonic saline solution Kellgren could evoke pain in all three areas. Moreover by injecting the interspinous ligaments of the vertebral column at each segmental level he induced pain in the extremities and the trunk and the pain followed a specific segmental pattern no different from that obtained by stimulating the individual muscles supplied by the same spinal segments (Figs 263 and 264).

Deep segmental pain according to the above investigators is independent of the superficial or cutaneous form described by Foerster and by Head. Their segmental pattern is similar but discloses some differences particularly in the extremities. Segmental sensory areas of the superficial system were designated "dermatomes" by Head and skin areas by Sherrington. Dermatomes extend to a lower level in the extremities than the pattern of deep segmental pain (compare Figures 265, 266 and 267 with Figure 264).

From these observations, Kellgren concluded that the segmental pattern of referred pain following stimulation of a muscle is determined by the nerve root or roots supplying the muscle. The pattern will include other muscles supplied by the same nerve root or roots.

For example the ninth intercostal nerve

roots. These areas (designated dermatome) provide a useful guide in determining the nerve root responsible for pain in a specific area of the extremity (Figs. 265, 266 and 267).

In the extremities, in contrast with the trunk, there is considerable overlap of cutaneous distribution of the individual root. This adds some difficulty in identifying the precise root affected. Diagnostic difficulties may be further increased by pain in only a small part of a specific dermatome. Eaton explains this by the theory of incomplete involvement of the nerve root. Theoretically, pain is referred to the entire cutaneous distribution of a nerve root when all the sensory fibers comprising the root are stimulated. However, if only a few of the fibers are involved, pain is projected only to that portion of the dermatome supplied by the affected sensory fiber.

This mechanism affords an explanation for the presence of isolated, localized areas of pain in the shoulder region, the arm or the fingers, resulting from compression of a sensory nerve root by protrusion of a disk in the cervical region. For example, in one patient with a protrusion of a disk between the fifth and the sixth cervical vertebrae which was proved subsequently, the syndrome was initiated by excruciating pain in the index finger with no other objective or subjective sensory manifestation.

Eaton cites two other features peculiar to radicular pain: (1) increased intra-abdominal or intrathoracic pressure tends to intensify or reproduce the pain, and (2) stretching of an affected root accentuates the pain.

The investigator demonstrated by clinical experiments that increased intra-abdominal and intrathoracic pressure resulting from such acts as sneezing, coughing, and straining produces distention of the epidural veins lying in loose areolar tissues in the epidural space between the dura mater and the spinal canal, consisting of the vertebral osseous elements, the intervertebral disks and the connecting spinal ligaments. The epidural veins are continuous with intervertebral

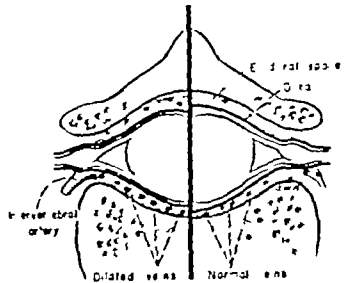


FIG. 265. Cross section of the spinal cord and epidural space during straining maneuvers on the left and before straining on the right. (Modified from Eaton, L. M. JAMA 117: 1433.)

veins which in turn flow into the abdominal and the thoracic vein. It is obvious that collapse or even compression of these large veins by increased intra-abdominal or intrathoracic pressure will cause enlargement of the epidural veins.

According to Eaton, this distention results in displacing the dura toward the spinal cord, thereby making traction on the nerve root. This observer does not believe that traction or compression of normal nerve roots by the distended epidural veins can produce pain, although traction on traumatized or diseased roots may evoke radiating pain. He further demonstrated that increased pressure in the cerebrospinal fluid plays no part in the production or the accentuation of root pain (Fig. 265).

Acts which tend to stretch injured nerve roots usually reproduce or intensify radicular pain. Eaton points out that the nerve roots are firmly fixed peripherally after they emerge from the intervertebral foramina. During maneuvers such as bending of the head far forward, forward bending of the trunk with knees extended, and Lasègue's maneuver (with the thigh flexed on the abdomen, the leg is extended), the nerve roots are stretched. While no pain is elicited under normal conditions, stretching of trau-

an associated phenomenon of referred pain and is not always indicative of a pathologic process in the underlying tissues

Pain and tenderness at the deltoid tuberosity and in the deltoid muscle are common findings in such disorders involving the supraspinatus region of the musculotendinous cuff as calcareous tendinitis and tears of the cuff. Lesions involving the biceps tendon (bicipital tenosynovitis) are characterized by pain projected to the belly of the biceps brachii muscle and by tenderness and soreness discernible by palpation and pressure

It becomes obvious from the afore mentioned experimental and clinical data that false localization of pain in the shoulder region may result from irritation of distant mesodermal tissues innervated by the same spinal segments as the areas to which the pain is projected. As demonstrated by Kellgren injection of the fifth cervical interspinous ligament innervated by the fifth cervical nerve produced pain in the region of the deltoid supplied by the same nerve. Shoulder and arm pain associated with diseases of the heart and the gallbladder is common knowledge

Kellgren and Lewis have produced pain which exhibits a segmental pattern and is associated simultaneously with cutaneous deep tenderness and muscle rigidity by stimulating somatic structures. Such phenomena resemble closely those produced by stimulation or diseases of the viscera. The observers are of the opinion that the objective manifestations of both somatic and visceral stimulation are indistinguishable. Lewis records "a manifest and simple explanation for these remarkable resemblances which include the pain phenomena and the associated rigidities and tenderness is the existence of a common though complex mechanism which is stirred into activity by afferent impulses derived from either deep-lying somatic structures or from disturbances of a viscus

Although the mechanism of pain referred from somatic structures or viscera is still obscure and the source of much controversy

and conjecture and in spite of the fact that such pain is not pathognomonic of any specific lesion the characteristics of the pain may provide helpful diagnostic clues.

Skin and deep pain possess distinguishing features. Clinical and experimental data lead one to conclude that they are two distinct forms of sensation (Lewis). Cutaneous pain is characterized by quick reflex phenomena either fleeting or prolonged, of rather even intensity, depending upon the type of stimulation and accurately localized to specific areas. Deep pain originating from muscle, fascia and periosteum is best described as being dull and boring. It is rather diffuse in nature and difficult to localize to a specific region. It is often associated with sweating, nausea, vomiting, drop in blood pressure. If the pain is of great intensity, the patient may even collapse. Patients often refer to this vasovagal phenomenon as sickening. The above features never are associated with pain of cutaneous origin. Some observers believe that the diffuse character of the pain is the result of intra-segmental diffusion, the extent of area involved depending upon the intensity of the pain.

Inman and Saunders postulate that "lesions of mesodermal structures bring about through some obscure reflex mechanism, radiation of pain along the pathways corresponding to the approximate segmental innervation of the periosteum, ligamentous structures and attachments of muscles.

As previously noted these pathways were designated sclerotomes and are in no way related to the dermatomes of peripheral nerves.

Pain is often projected to the region of the shoulder and the arm by compression of one of the cervical sensory nerve roots. Radicular or root pain exhibits characteristic features which are helpful diagnostic aids. Pain is referred to the cutaneous area supplied by the affected root thereby disclosing a definite segmental pattern. Sherrington and more recently Foerster have deciphered and charted the cutaneous pattern of distribution of the sensory nerve

provide a bony enclosure (pinal canal) for the 8 cervical segments and a portion of the first thoracic segment of the pinal cord. No disk exists between the occiput and the atlas or between the atlas and the axis. The first cervical disk is situated between the second and the third cervical vertebrae. Only a small exit is found for emergence of the second cervical root between the atlas and the axis. Only 6 well formed bony foramina are demonstrable in the cervical region. The first is situated between the second and the third cervical vertebrae. The first cervical nerve takes its exit between the occiput and the atlas.

It is obvious that confusion may arise in referring to specific levels in the cervical region. For example the fourth disk is located between the fifth and the sixth cervical vertebrae which form the fourth intervertebral foramina which in turn is traversed by the sixth cervical nerve. Figure 270 discloses that the cervical nerves emerge from intervertebral foramina above their respective vertebrae. For example the sixth cervical nerve traverses the foramen above the sixth cervical vertebra. To avoid confusion in referring to lesions of the intervertebral disks it is best to designate the specific nerve involved or to identify the disk by naming the specific vertebrae above and below—for example the intervertebral disk between the fifth and the sixth cervical vertebrae.

As mentioned above, there are 6 intervertebral foramina from which emerge the last six cervical nerves. Each foramen is ovoid in shape with a large vertical and a smaller horizontal diameter. Ample room is provided for the cervical nerve roots which occupy roughly half of the available space in the foramen. This last anatomic feature is of special clinical significance. Some observers maintain that narrowing of the intervertebral disk is followed by hypertrophic arthritis that narrowing of the intervertebral disk may reduce the height of the vertical diameter of the foramen and compress the nerve roots (Turner and Oppenheimer).

According to Semmes and Murphey narrowing of the foramen subsequent to thinning of the intervertebral disk does not cause nerve root pressure. They contend that even with total obliteration of the intervertebral disk space the foramen is spacious enough to accommodate the cervical nerve and root without causing pressure. However destruction of adjacent surfaces of the vertebrae in addition to thinning of the intervertebral disk may narrow the vertical diameter of the foramen sufficiently to cause pressure on the nerve root. Lesions which reduce the horizontal diameter of the foramina are more apt to cause compression of the nerve root. This explains the compression syndrome that may ensue when a small lateral protrusion of the intervertebral disk occurs. Although there is evidence substantiating the observation that hypertrophic arthritis *per se* is not apt to reduce the size of the foramina sufficiently to produce radicular pain one must realize that associated inflammatory processes (affecting the ligamentous structures and the capsular tissues surrounding the foramen) may reduce the lumen of the foramen sufficiently to cause nerve root pressure.

Moreover in the light of the work of Kellgren radicular pain to the shoulder and the arm may result from irritation of the deep ligaments and the capsular tissues in the cervical region by a process such as hypertrophic arthritis. He was able to produce segmental pain in the arm by stimulating the sixth and the seventh interspinous ligaments.

The seven cervical vertebrae form a smooth lordotic curve whose apex is roughly at the fifth or the sixth vertebra. At the apex of the curve, the intervertebral foramina (those between the fifth and the sixth and the sixth and the seventh vertebrae) are narrower than those above and below (Semmes and Murphey). The two intervertebral disks above—particularly the one between the sixth and the seventh cervical vertebrae—are vulnerable to stress and strains because of their greater mobility.

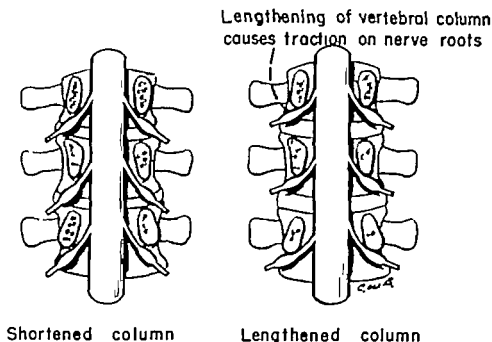


FIG 269 Diagram showing effect of lengthening of the vertebral column on the nerve roots. Note that the spinal cord and the nerve roots are stretched.

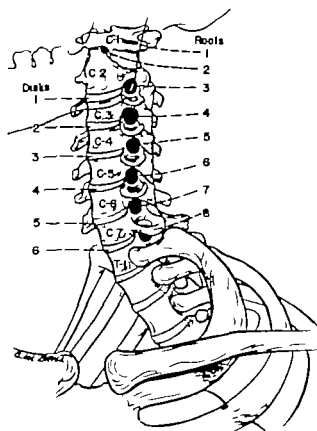


FIG 270 Tracing made from a roentgenogram of the cervical and upper thoracic spine showing the intervertebral foramina, intervertebral disks and vertebral bodies.

matized nerve roots causes or accentuates radicular pain. Often root pain is reproduced or intensified during sleep and is relieved on getting out of bed.

Eaton contends that the weight of the body in an upright position compresses the intervertebral disks, thereby reducing the height of the spinal column—as compared with its length in the horizontal position during sleep. In the latter position the increased length is secondary to the increase in the vertical height of the intervertebral disks that occurs when the disks are relieved of the weight of the body. The increase in length of the spinal column stretches the spinal cord and the nerve roots. Under normal conditions such increased tension in the cord and the roots is within normal physiologic limits and produces no pain; however stretching of diseased or traumatized roots may provoke or accentuate radicular pain (Fig 269).

ANATOMIC CONSIDERATIONS

CERVICAL SEGMENT OF THE SPINAL COLUMN

This segment comprises 7 cervical vertebrae and 6 intervertebral disks which

cervical and first thoracic nerve. The seventh cervical nerve supplies the ax of the limb but also gives off sensory nerve fibers which innervate such deep structure as periosteum, ligaments and muscle.

Variation of the pinal segments comprising the brachial plexus are encountered the most common being designated the prefixed and the postfixed plexuses. In a prefixed plexus the entire plexus is at a higher level, a large portion of the anterior primary ramus of the fourth cervical nerve goes into its formation but receives few fibers from the first thoracic nerve. A postfixed plexus occupies a lower level. It receives a large contribution from the anterior primary ramus of the second thoracic nerve but receives no fibers from the fourth and only a few from the fifth cervical nerves.

As will be shown subsequently both prefixed and postfixed plexuses are at times responsible for radiating pain in the shoulder and the arm especially if such anomalies of the thoracic outlet exist as a cervical rib or abnormal first thoracic rib. As previously recorded the cutaneous distribution of the sensory fibers of a pinal nerve exhibits a segmental pattern designated dermatome. There is however great overlapping of the adjacent dermatomes and it may be impossible to identify the sensory root affected by abnormal sensory manifestations in a specific area.

Ordinarily the anterior primary rami of the fifth and the sixth cervical nerves form the upper trunk, that of the seventh continues as the middle trunk while the rami of the eighth cervical and first thoracic nerves constitute the lower trunk. Each trunk divides into an anterior and a posterior division from which are derived the three cords of the brachial plexus. The lateral cord consists of the anterior divisions of the upper and the middle trunk, the posterior cord embodies the posterior division of all three trunks and the anterior division of the lower trunk continues as the medial cord. From the three cords arise the major peripheral nerves of the upper extremity.

The lateral cord gives origin to the musculocutaneous nerve and the lateral head of the median nerve, also to the lateral anterior thoracic nerve. The posterior cord gives origin to the axillary and the radial nerve and to the two ulnar-carpular and the thoracodorsal nerve. From the medial cord are derived the medial head of the median nerve and the ulnar nerve, also the medial anterior thoracic nerve and the medial cutaneous nerves of the forearm and the arm (Fig. 243). Rearrangement of the nerve fiber comprising the brachial plexus take place in the division of the plexus. In general those fibers which will supply the ventral part of the extremity reach their destination through the anterior division, and those supplying the dorsal parts through the posterior division. Table 2 gives the segmental supply of the skeletal muscles of the neck, the shoulder and the upper arm. Figures 265, 266 and 267 depict the segmental pattern of sensory supply of the skin of the upper limbs and the upper portion of the trunk.

Sympathetic innervation to the upper extremity arises in the lateral horn cells of the pinal segments T3 to T6 inclusive. Some investigators believe that T2 and T7 may also contribute fibers to the brachial plexus. Preganglionic fibers which arise in the above pinal segments leave the pinal cord via the corresponding anterior roots, traverse the primary anterior rami and reach the thoracic sympathetic chain as white rami communicantes. They then ascend to the stellate and middle cervical ganglia where they synapse with postganglionic fibers. The postganglionic fibers as gray rami communicantes reach the anterior primary rami of the brachial plexus and then the periphery via the peripheral nerves. They supply the blood vessels, the sweat glands and the pilo-erector muscles (Fig. 272).

The preganglionic fibers destined to innervate the ocular structures traverse the primary rami of T1 and T2 and possibly T3. They enter the sympathetic chain via

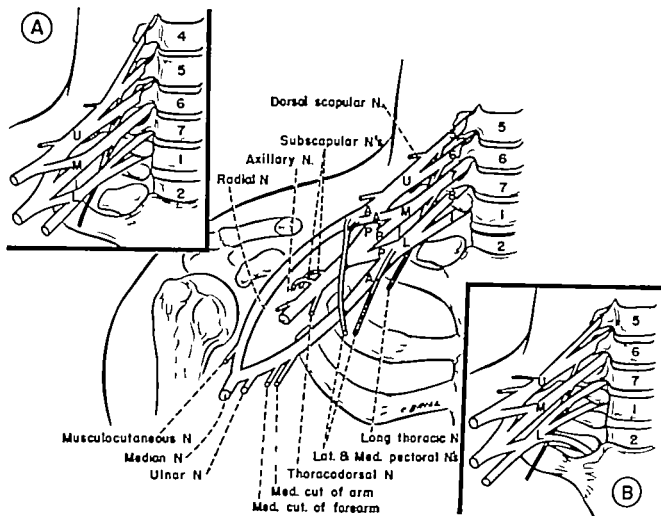


FIG. 271 Brachial plexus and the most common variations (A) Prefixed plexus. (B) Postfixed plexus. (Haymaker and Woodhall *Peripheral Nerve Injuries* Philadelphia, Saunders p 129 Redrawn with supplementations.)

and because they comprise the transitional area between the freely movable upper cervical spine and the fixed thoracic spine (Bradford and Spurling). Because of the afore mentioned anatomic features and the functional demands made on this lower cervical region it is the most frequent site of pathologic disorders especially those of mechanical nature. Degeneration with or without protrusion of the disks between the fifth and the sixth and the sixth and the seventh cervical vertebrae are common lesions.

BRACHIAL PLEXUS

Spinal nerves supplying the extremities differ from those supplying the trunk in that they form plexuses. In the early development of the upper extremities a specific

number of metameres migrated distally to form the limb buds. The respective spinal nerves grow into the limb bud; their anterior primary rami form plexuses in which the nerve fibers are grouped so that the upper spinal nerves of the plexus supply the preaxial border of the limb and the lower spinal nerves supply the postaxial border. The central portion of the plexus is carried distally the full length of the limb and supplies the axis of the extremity.

The brachial plexus comprises the anterior primary rami of the fifth, sixth, seventh and eighth cervical and the first thoracic nerves. In accordance with the afore mentioned scheme the preaxial or radial surface of the limb is supplied by the fifth and the sixth cervical nerves and the postaxial or ulnar surface by the eighth

the white rami communicantes and then extend upward through the cervical ganglia to reach the superior ganglion where they synapse with postganglionic fibers which reach the ocular arteries. Walsh, Jackson and Wyburn-Mason point out that the only situation in which the sympathetic fibers to head, neck, forequarter and upper limbs are gathered together and capable of simultaneous involvement by a single lesion is in the stellate ganglion."

TOPOGRAPHIC AND VARIATIONAL ANATOMY OF THE SUPRA CLAVICULAR REGION

A knowledge of the topographic anatomy of the components of the brachial plexuses and of the variational anatomy of this region is essential to understand the mechanism of the different compression syndromes described. All have in common pain projected to the shoulder region and the arm. The undivided primary anterior rami take their exit from the intervertebral foramina and traverse grooves directed downward and laterally in the anterosuperior surface of the transverse processes of the cervical vertebrae. In its respective groove each root lies directly behind a slip of origin of the scalenus anticus muscle which arises from the anterior tip of the transverse process above. For example the root of the fourth cervical nerve is situated between the slip of origin of the scalenus muscle from the tip of the transverse process of the third cervical and the groove in which it lies in the transverse process of the fourth cervical. Thus the cervical nerve roots may be compressed by the slip of scalenus anticus muscle (Swank and Simeone).

The scalenus medius muscle lies behind the cervical roots. It arises from the posterior tubercle of the transverse processes of the second to the sixth cervical vertebrae. Both anticus and medius scalene muscles insert into the first thoracic rib. Between them lies the subclavian artery and the roots of the brachial plexus whose lower elements are situated immediately above the

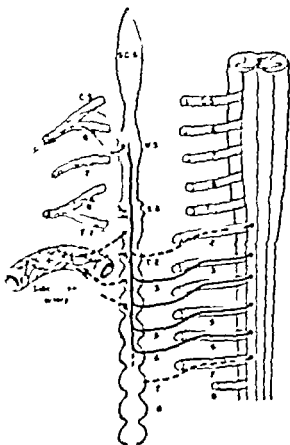


FIG. 272 Diagrammatic representation of the sympathetic innervation of the upper extremity. Preganglionic fibers take origin from spinal segments T₃ to T₄ (solid lines) and perhaps from T₅ to T₇ (dotted lines). At the point of emergence from anterior primary rami they form white rami communicantes. Postganglionic neurons (indicated by broken lines) extend to components of the brachial plexus and to the subclavian artery as gray rami communicantes. SCG, superior cervical sympathetic ganglion; MG, middle cervical sympathetic ganglion; SG, stellate ganglion. (Haymaker and Woodhall: *Peripheral Nerve Injuries*, Philadelphia: Saunders, p. 11.)

artery. According to Swank and Simeone the lower portions of the two muscles are relatively rigid and inelastic by virtue of their attachment to the first rib.

This fixed position may be increased by a prominent seventh cervical transverse process or a cervical rib. It is conceivable from the above topographic anatomy that both upper and lower cervical roots are vulnerable to compression by hypertrophy or spasticity of the scalene muscles. Swank

SPINAL SEGMENTS								
C1	C2	C3	C4	C5	C6	C7	C8	T1
Sternomastoid								
	Trapezius							
		Levator scapulae						
			Teres minor					
				Supra- spinatus				
				Rhomboids				
				Infraspinatus				
				Deltoid				
				Teres major				
				Biceps				
				Brachialis				
				Serratus anterior				
				Subscapularis				
				Pectoralis major				
					Pectoralis minor			
					Coraco- brachialis			
					Latissimus dorsi			
						Anconeus		
						Triceps		

TABLE 2 Segmental supply of skeletal muscles of neck, shoulder and upper arm. The asterisks indicate a supply also from the accessory nerve. A line bisecting segment indicates that the muscle received a minor innervation from the segment. (Haymaker and Woodhall *Peripheral Nerve Injuries*, Philadelphia, Saunders p 26—Modification of Bing)

slightly forward but are restrained by the insertion of the scalenus anticus muscle, which prevents the neurovascular bundle from rolling forward (Fig 273)

As the subclavian artery and the trunks of the plexus continue laterally and downward they come to lie between the clavicle and the first thoracic rib. At the lateral border of the first rib the subclavian artery becomes the axillary artery and it together with the three cords passes posterior to the pectoralis minor muscle just below the coracoid process.

Numerous bony abnormalities occur in the region of the thoracic operculum which may be responsible for compression syndromes. Walshe, Jackson and Wyburn Mason believe that the clavicle and the abnormal rib either of cervical or thoracic origin form a vise in which the subclavian vessels and trunks of the plexus are compressed repeatedly.

Lesions of neurogenic origin responsible for pain in the neck, the shoulder and the arm are best grouped into three categories.

- 1 Lesions situated within spinal cord and spinal canal
- 2 Lesions associated with pathologic disorders of the vertebral column
- 3 Lesions situated outside the vertebral column

The important lesions situated within the spinal cord and the spinal canal to be considered in this group are

- 1 Neoplasms of the cervical spinal cord and nerve roots
- 2 Syringomyelia
- 3 Hypertrophic cervical pachymeningitis
- 4 Platybasia
- 5 Herpes zoster
- 6 Inflammatory lesions

Neoplasms within the spinal canal may be situated extradurally or intradurally, the latter group may be subdivided into intramedullary and extramedullary tumors. According to Davis 75 per cent of all spinal cord tumors are extramedullary and 75 per cent of these are intradural. The subjective and the objective neurologic features and

the clinical course depend upon the size and the location of the tumor.

Intramedullary tumors of the cervical segments of the spinal cords are relatively rare when present pain may be referred to the shoulders and the upper extremities and it points to involvement of the sensory tracts in this region. As a rule pain in central lesions is widespread and projected to greater areas as to the trunk and even to the lower extremities. The central location of the lesion may be further disclosed by the presence of muscular weakness, spasticity, hyperactive tendon reflexes and extensor plantar phenomena (Babinski)—all indicating involvement of the pyramidal tracts. Altered sensory phenomena such as loss of pain and temperature sense disclosing involvement of the spinothalamic pathways and impairment of position and vibratory sense pointing to damage to the posterior columns are all supportive evidence of an intramedullary lesion.

Extramedullary but intraspinal tumors frequently give rise to radicular pain referred to the shoulder and the arm. This is especially true of lesions below the fourth cervical segment. They arise from the coverings of the spinal nerves and cord are usually discrete encapsulated tumors which are readily accessible and as a rule their removal is followed by gratifying results. Radicular pain projected to the shoulder or the arm without the neurologic manifestations may be the only symptom of extramedullary neoplasm in the cervical spinal cord or nerve roots.

Eaton records three characteristics of radicular pain which are helpful in differential diagnosis. The first and most significant is its segmental nature. In involvement of the eighth cervical nerve root pain is referred to inner side of the arm, the forearm, the hand and the fifth finger and occasionally to the fourth and the fifth fingers. Involvement of the seventh nerve root projects pain to the index finger and at times to the third finger and the thumb while involvement of the fifth and the sixth nerve

and Simeone have described two scalenus anticus syndromes superior and inferior types depending upon the level of the plexuses affected

The elements of the brachial plexus distal to the scalene muscles and occupying the supraclavicular fossa comprise the trunks. The divisions lie behind the middle third of the clavicle and the cords are located in the axilla commencing just beyond the outer

border of the first thoracic rib. The first thoracic nerve root and, at times, the second (particularly when a postfixed plexus is present) must arch upward and over the first thoracic rib to reach the next higher root. It is situated just lateral to and slightly above the subclavian artery, which separates the nerve root from the posterior border of the scalenus anticus muscle. Both artery and plexus tend to be directed

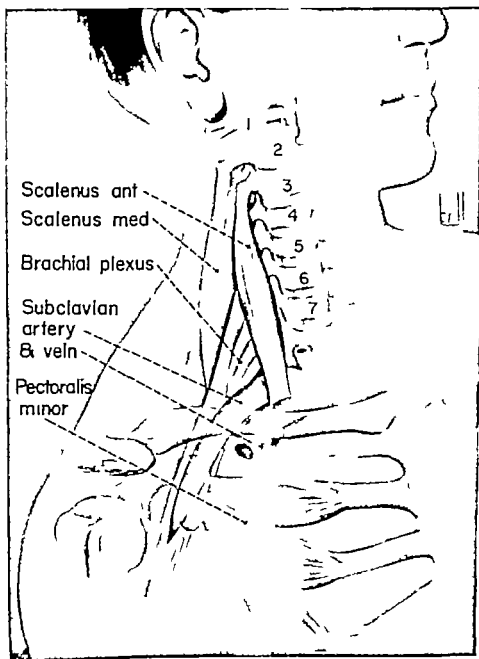


FIG 273 Drawing of anatomic specimen showing the topographic anatomy of the brachial plexus and the large vessels—particularly in relation to the first thoracic rib and clavicle the scalenus anticus muscle and pectoralis minor muscle and the coracoid process.

slightly forward but are restrained by the insertion of the scalenus anticus muscle, which prevents the neurovascular bundle from rolling forward (fig 273)

As the subclavian artery and the trunks of the plexus continue laterally and downward they come to lie between the clavicle and the first thoracic rib. At the lateral border of the first rib the subclavian artery becomes the axillary artery and it together with the three cords passes posterior to the pectoralis minor muscle just below the coracoid process.

Numerous bony abnormalities occur in the region of the thoracic operculum, which may be responsible for compression syndromes. Walsh, Jackson and Wyburn Mason believe that the clavicle and the abnormal rib either of cervical or thoracic origin form a vise in which the subclavian vessels and trunks of the plexus are compressed repeatedly.

Lesions of neurogenic origin responsible for pain in the neck, the shoulder and the arm are best grouped into three categories.

- 1 Lesions situated within spinal cord and spinal canal
- 2 Lesions associated with pathologic disorders of the vertebral column
- 3 Lesions situated outside the vertebral column

The important lesions situated within the spinal cord and the spinal canal to be considered in this group are

- 1 Neoplasms of the cervical spinal cord and nerve roots
- 2 Syringomyelia
- 3 Hypertrophic cervical pachymeningitis
- 4 Platybasia
- 5 Herpes zoster
- 6 Inflammatory lesions

Neoplasms within the spinal canal may be situated extradurally or intradurally. The latter group may be subdivided into intramedullary and extramedullary tumors. According to Davis 75 per cent of all spinal cord tumors are extramedullary and 75 per cent of these are intradural. The subjective and the objective neurologic features and

the clinical course depend upon the size and the location of the tumor.

Intramedullary tumors of the cervical segments of the spinal cord are relatively rare when present pain may be referred to the shoulders and the upper extremities and it points to involvement of the sensory tracts in this region. As a rule pain in central lesions is widespread and projected to greater areas as to the trunk and even to the lower extremities. The central location of the lesion may be further disclosed by the presence of muscular weakness, spasticity, hyperactive tendon reflexes and extensor plantar phenomena (Babinski)—all indicating involvement of the pyramidal tracts. Altered sensory phenomena such as loss of pain and temperature sense disclosing involvement of the spinothalamic pathways and impairment of position and vibratory sense pointing to damage to the posterior columns are all supportive evidence of an intramedullary lesion.

Extramedullary but intra-spinal tumors frequently give rise to radicular pain referred to the shoulder and the arm. This is especially true of lesions below the fourth cervical segment. They arise from the coverings of the spinal nerves and cord are usually discrete encapsulated tumors which are readily accessible and as a rule their removal is followed by gratifying results. Radicular pain projected to the shoulder or the arm without the neurologic manifestations may be the only symptom of extramedullary neoplasm in the cervical spinal cord or nerve roots.

Eaton records three characteristics of radicular pain which are helpful in differential diagnosis. The first and most significant is its segmental nature. In involvement of the eighth cervical nerve root pain is referred to inner side of the arm, the forearm, the hand and the fifth finger and occasionally to the fourth and the fifth fingers. Involvement of the seventh nerve root projects pain to the index finger and at times to the third finger and the thumb while involvement of the fifth and the sixth nerve

roots is responsible for pain in the shoulder and outer (radial) aspects of the forearm and the thumb and frequently the index finger. The second characteristic is accentuation of the pain by increased intra-thoracic and such intra-abdominal tension as is produced by coughing, sneezing and straining. (The mechanism of this phenomenon has been described previously.)

Finally, the third is increase in the intensity of radicular pain by maneuvers which stretch the affected nerve roots, such as flexing the head until the chin rests on the chest wall or downward traction on the arm. This explains the severe nocturnal pain in these cases due to elongation of the spinal column when in a horizontal position, thereby stretching the involved nerve root. Relief from pain is attained by assuming an upright position. The root pain may be dull and constant in nature but more often it is sudden, sharp and lancinating. It may be accentuated or reproduced by certain motions of the spinal column.

With increase in the size of the tumor pressure may be made on the spinal cords resulting in involvement of the sensory and the motor pathways and producing signs and symptoms similar to those described for intramedullary tumors. In brief, impairment of the spinothalamic tracts produces loss of pain and temperature sense. Involvement of the posterior columns is characterized by disturbance of the position and vibratory sense. Pyramidal tract involvement is associated with muscle weakness, paresis, hyperactive tendon reflexes and abnormal extensor/plantar phenomena. A Brown-Séquard syndrome may result from a tumor which makes lateral pressure on the spinal cord.

Other aids helpful in establishing a diagnosis of a neoplasm in the cervical spinal cord or nerve roots are:

1. Radiographic study is made on stereoscopic, anteroposterior, lateral and oblique views of the cervicothoracic spine. Destruction of the pedicles or of the vertebral body may be demonstrable particularly in the presence of an extramedullary tumor.

Widening of the interpedicular space with or without bone destruction is a significant finding indicative of a neoplasm in the involved area.

2. Examination of the cerebrospinal fluid usually reveals an elevated protein level. The fluid is often yellow in color (xanthochromia).

3. Manometric study gives valuable information to determine the presence of a partial or complete spinal subarachnoid block. The Queckenstedt test and the method of Grant and Cone (who employ a blood pressure cuff around the neck to obtain varying degrees of compression of the jugular vein) are the most valuable manometric tests.

4. Myelography may localize the lesion as well as establish the diagnosis.

SYRINGOMYELIA

This lesion may produce signs and symptoms similar to those of intramedullary tumors of the cervical spinal cord and occasionally mimics those produced by cervical rib. Although pain is not a cardinal symptom of this disease, occasional deep, boring or burning pain in the shoulder and arm and stiffness of the neck may be prominent features of the clinical picture.

This process usually involves the lower cervical and upper thoracic spinal cord. Briefly, the process comprises proliferation of the glial cells in the region of the central canal (designated central gliosis), cystic degeneration and cavitation of the cord. The process is limited at first to the gray matter of the cord; later it involves the white matter. It tends to extend upward and downward anteriorly and posteriorly, only rarely does it extend laterally across the cord. It becomes apparent that the signs and symptoms manifested are dependent upon the area of the cord affected. In the early stages it progresses with relative rapidity, however, quiescent periods are characteristic of the disease. Other frequent developmental defects are platybasia, Klippel-Feil deformity and the cervical rib.

Syringomyelia is encountered most frequently in the second and the third decades, usually it progresses slowly and remains in a quiescent state for many years becoming progressive again. Hemorrhage into the cystic tissue may reactivate the process.

In the early stages when the process is confined to the gray matter about the central canal loss of pain and temperature sense are the most significant symptoms, and it involves only the pinothalamic tract on one side. Inasmuch as the lower cervical and the upper thoracic segments of the spinal cord are the regions most commonly affected the symptoms are projected to the neck, the shoulder, the arms and the upper thoracic regions. It is common knowledge that syringomyelic patients unknowingly sustain burns of the fingers from lighted cigarettes or hot water. This may be the first clue of loss of pain and temperature sense. The first indication of the disorder may be numbness of the fingers. Loss of pain and temperature is always segmental in nature and found on both sides of the body below the level of the lesion.

It is characteristic of this disease that touch, deep pressure and position senses are not impaired because the posterior columns are seldom involved. Occasionally the posterior columns do become affected and ataxia develops. The cells of the anterior horn are involved with extension of the pathologic process resulting in atrophy, weakness and paralysis of muscles. Motor disturbances also disclose a segmental arrangement. Weakness of the muscles supplied by the ulnar nerve and atrophy of the hypothenar eminence are usually the first motor manifestations. Similar involvement of the muscles of the shoulder and the pectoral regions occurs with further extension of the process in the spinal cord. Motor manifestations are at first unilateral, later both extremities are affected as the process extends to the opposite side of the cord. Occasionally the lesion destroys the pyramidal tracts resulting in paralysis or weak-

ness of muscle, pallidity, hyperactive tendon reflexes and extensor plantar phenomena.

Implication of the cells of origin of the sympathetic fibers situated in the lateral horns of the gray matter is exhibited by vasomotor disorders in the upper extremity. The hands are moist, cool and slightly edematous. Sometimes Horner's syndrome is present resulting from involvement of the eighth cervical and the first thoracic segment. Trophic changes such as indolent ulcers of the fingers are frequently encountered following burns. Rarely neurotrophic joints (Charcot joints) develop in syringomyelia. The shoulder and elbow joints are most commonly affected.

HYPERTROPHIC CERVICAL PACHYMENINGITIS

This lesion is essentially a chronic inflammatory process, involving the dura mater of the cervical segment of the spinal cord and the cervical nerve roots. Many cases are syphilitic but most are nonspecific in origin. The lesion is not common and may be confused with syringomyelia or intramedullary tumors in the cervical region of the spinal cord. Pain in the neck, the shoulders and the arms is a prominent factor of the disease. The pain may be severe, is segmental in nature and is accentuated by movements of the cervical spine. This is followed by progressive muscular weakness, atrophy and fibrillation of muscles innervated by the affected cervical nerve roots. The muscles of the hands and the arms are most markedly affected. Extension of the inflammatory process may involve the spinal cord or produce compression of the cord resulting in weakness, spasticity and sensory disturbances in the lower extremity. Varying degrees of anatomic block may exist depending upon the extent of the inflammatory process and the amount of compression of the spinal cord. Lesions of syphilitic origin will exhibit positive serology of the blood and the spinal fluid and the diagnosis may be substantiated further by the pres-

roots is responsible for pain in the shoulder and outer (radial) aspects of the forearm and the thumb, and frequently the index finger. The second characteristic is accentuation of the pain by increased intra-thoracic and such intra-abdominal tension as is produced by coughing, sneezing and straining (The mechanism of this phenomenon has been described previously.)

Finally, the third is increase in the intensity of radicular pain by maneuvers which stretch the affected nerve roots, such as flexing the head until the chin rests on the chest wall or downward traction on the arm. This explains the severe nocturnal pain in these cases due to elongation of the spinal column when in a horizontal position, thereby stretching the involved nerve root. Relief from pain is attained by assuming an upright position. The root pain may be dull and constant in nature but more often it is sudden sharp and lancinating. It may be accentuated or reproduced by certain motions of the spinal column.

With increase in the size of the tumor pressure may be made on the spinal cords resulting in involvement of the sensory and the motor pathways and producing signs and symptoms similar to those described for intramedullary tumors. In brief impairment of the spinothalamic tracts produces loss of pain and temperature sense. Involvement of the posterior columns is characterized by disturbance of the position and vibratory sense. Pyramidal tract involvement is associated with muscle weakness, paresis, hyperactive tendon reflexes and abnormal extensor plantar phenomena. A Brown Sèquard syndrome may result from a tumor which makes lateral pressure on the spinal cord.

Other aids helpful in establishing a diagnosis of a neoplasm in the cervical spinal cord or nerve roots are

1. Radiographic study is made on stereoscopic, anteroposterior, lateral and oblique views of the cervicothoracic spine. Destruction of the pedicles or of the vertebral body may be demonstrable particularly in the presence of an extramedullary tumor.

Widening of the interpedicular space with or without bone destruction is a significant finding indicative of a neoplasm in the involved area.

2. Examination of the cerebrospinal fluid usually reveals an elevated protein level. The fluid is often yellow in color (xanthochromia).

3. Manometric study gives valuable information to determine the presence of a partial or complete spinal subarachnoid block. The Queckenstedt test and the method of Grant and Cone (who employ a blood pressure cuff around the neck to obtain varying degrees of compression of the jugular vein) are the most valuable manometric tests.

4. Myelography may localize the lesion as well as establish the diagnosis.

SYRINGOMYELIA

This lesion may produce signs and symptoms similar to those of intramedullary tumors of the cervical spinal cord and occasionally mimics those produced by cervical rib. Although pain is not a cardinal symptom of this disease, occasional deep boring or burning pain in the shoulder and arm and stiffness of the neck may be prominent features of the clinical picture.

This process usually involves the lower cervical and upper thoracic spinal cord. Briefly, the process comprises proliferation of the glial cells in the region of the central canal (designated central gliosis), cystic degeneration and cavitation of the cord. The process is limited at first to the gray matter of the cord; later it involves the white matter. It tends to extend upward and downward anteriorly and posteriorly; only rarely does it extend laterally across the cord. It becomes apparent that the signs and symptoms manifested are dependent upon the area of the cord affected. In the early stages it progresses with relative rapidity; however, quiescent periods are characteristic of the disease. Other frequent developmental defects are platybasia, Klippel-Feil deformity and the cervical rib.

joint caused by pain or muscular involvement results in a frozen shoulder which in itself is a markedly disabling malady

INFLAMMATORY LESIONS

Epidural abscess may be responsible for segmental pain in the shoulder and the arm. It may be a metastatic lesion from a pyogenic focus outside of the central nervous system or it may be a secondary lesion derived from pyogenic organisms in the blood stream. The signs and symptoms depend upon the number of cervical and upper thoracic nerve roots implicated and the degree of pressure made by the extradural inflammatory mass on the spinal cord. In general, radicular pain is present, also, abnormal sensory and motor manifestations below the level of the lesion. Dysfunction of the spinal cord, such as progressive paraplegia, may progress rapidly due to extension of the inflammatory mass which compresses the cord. The appearance of flaccid paralysis is an ominous sign.

As a rule, diagnosis is facilitated by such associated systemic reactions as spiking temperature range, chills, prostration, high leukocytosis and low red blood-cell count. A frank suppurative area elsewhere may be a diagnostic clue. Studies of the cerebrospinal fluid disclose a high cell count and an increased protein level. Lumbar puncture usually reveals a complete manometric block of the subarachnoid space.

GUILLAIN BARRÉ SYNDROME

Pain in the upper extremity and the shoulder may be produced by acute inflammatory processes which involve the peripheral nerves, the roots or the cord singly or in combinations, often grouped under the all inclusive heading 'myelo-radiculo-neuritis' or the 'Guillain Barré Syndrome.' The syndrome is usually associated with some other systemic infection, particularly of the upper respiratory tract. The clinical course is variable. Generally there is root pain and varying degrees of motor and sensory impairment. The disease progresses



FIG. 274 Tuberculosis of the cervical spine in a male, 53 years old. The cardinal symptom was pain referred to the right shoulder and arm.

to a certain stage, remains static for a period and then recedes slowly. This last phase may persist for many months.

Rapidly ascending lesions with involvement of the brain which have terminated fatally have been recorded. Frequently cranial nerves are implicated. The cardinal motor features are progressive muscular weakness, flaccid paralysis, loss of deep tendon reflexes and tenderness along the course of the peripheral nerves. Characteristic of this syndrome is an increase in the protein content without appreciable change in the cells of the spinal fluid. The etiologic agent is obscure. Most evidence, however, points to a neurotropic virus.

LESIONS ASSOCIATED WITH PATHOLOGIC DISORDERS OF THE VERTEBRAL COLUMN

Radicular pain to the shoulder and the arm may result from stimulation of the cervical and the upper thoracic nerve roots or involvement of the deep mesodermal tis-

ence of other signs of syphilis of the central nervous system. However, nonspecific lesions will have a negative blood and spinal fluid serology. Excision of the posterior dural covering of the cervical spinal cord followed by irradiation, has been followed by marked relief of symptoms in some instances.

PLATYBASIA (BASILAR COMPRESSION)

Platybasia is a skeletal deformity characterized by malformation of the occipital bone and the upper cervical vertebrae. The foramen magnum is misshapen and narrow, the atlas, as a rule, is fused with the occipital bone, the clivus is displaced upward and the odontoid process compresses the medulla and the cervical segments of the spinal cord. Other associated anomalies are syringomyelia of the cervical segment of the spinal cord and the Klippel Feil deformity.

Gustafson and Oldberg are of the opinion that platybasia is responsible for the formation of cystic defects in the spinal cord similar to that of syringomyelia. List points out that neurologic manifestations associated with this skeletal deformity may result from mechanical compression of the brain stem or developmental defects of the central nervous system. Many etiologic factors responsible for platybasia have been cited including osteomalacia, rickets and osteitis deformans. However, most investigators believe that the lesion is a developmental defect. Trauma is not considered a primary causative factor, although it may influence the course of the disease.

The signs and symptoms and the character of the clinical course depend upon the degree of mechanical compression and the response of the arachnoid to irritation; both factors are capable of causing a partial or complete block of the subarachnoid space. It is apparent that the resultant syndrome is variable and may be confused with syringomyelia, intramedullary tumor and progressive spastic paralysis. In most instances roentgenographic examination dis-

closes the true nature of the lesion. Relief of symptoms may be attained by decompression of the foramen magnum.

HERPES ZOSTER

Herpes zoster is one of the less common causes of pain in the shoulder and the arm. It is an acute infectious disease whose etiologic agent has been definitely established as a filtrable virus. The seat of involvement is the posterior root ganglia, posterior roots and posterior gray horns of the spinal cord. In more severe forms, the inflammatory process may extend into the anterior roots and give rise to motor disorders. This disease is rarely encountered in the young age groups; it occurs most often after the fourth decade of life. Implication of the cervical and the upper thoracic sensory nerve roots produces radicular pain in the neck, the shoulder or the arm which is segmental in nature and has a pattern of cutaneous distribution depending upon the specific root or roots involved.

This disease is ushered in by hyperalgesia and hypersensitiveness in a specific dermatome; the pain may be intense and of a burning character. From 3 to 5 days after the onset, small discrete vesicles appear in the same segmental area corresponding to the affected root or roots. Pain is usually severe throughout the entire acute stage. A brown pigmentation of the area remains after the vesicles disappear and may persist for an indefinite period. Postherpetic intractable pain may ensue particularly in older individuals and it may persist for many years. Such distressing sequela often fails to respond to any form of therapy. In most cases, section of the affected roots fails to alleviate the pain.

Although rare segmental motor involvement may occur, it is a more serious problem in the upper extremity than elsewhere. The principal features are weakness or flaccid paralysis of muscles, atrophy and loss of deep reflexes. In many cases, prolonged loss of motion in the scapulohumeral

joint caused by pain or muscular involvement results in a frozen shoulder which in itself is a markedly disabling malady.

INFLAMMATORY LESIONS

Epidural abscess may be responsible for segmental pain in the shoulder and the arm. It may be a metastatic lesion from a pyogenic focus outside of the central nervous system or it may be a secondary lesion derived from pyogenic organisms in the blood stream. The signs and symptoms depend upon the number of cervical and upper thoracic nerve roots implicated and the degree of pressure made by the extradural inflammatory mass on the spinal cord. In general radicular pain is present, also abnormal sensory and motor manifestations below the level of the lesion. Dysfunction of the spinal cord, such as progressive paraplegia, may progress rapidly due to extension of the inflammatory mass which compresses the cord. The appearance of flaccid paralysis is an ominous sign.

As a rule diagnosis is facilitated by such associated systemic reactions as piking, temperature range, chills, prostration, high leukocytosis and low red blood-cell count. A frank suppurative area elsewhere may be a diagnostic clue. Studies of the cerebrospinal fluid disclose a high cell count and an increased protein level. Lumbar puncture usually reveals a complete manometric block of the subarachnoid space.

GUILLAIN-BARRÉ SYNDROME

Pain in the upper extremity and the shoulder may be produced by acute inflammatory processes which involve the peripheral nerves, the roots or the cord singly or in combinations, often grouped under the all-inclusive heading "myelo-radiculo-neuritis" or the Guillain-Barré Syndrome. The syndrome is usually associated with some other systemic infection, particularly of the upper respiratory tract. The clinical course is variable. Generally there is root pain and varying degrees of motor and sensory impairment. The disease progresses



FIG. 274. Tuberculosis of the cervical lymph node in a male 53 years old. The cardinal symptom was pain referred to the right shoulder and arm.

to a certain stage remain static for a period and then recedes slowly. This last phase may persist for many months.

Rapidly ascending lesions with involvement of the brain which have terminated fatally have been recorded. Frequently cranial nerves are implicated. The cardinal motor features are progressive muscular weakness, flaccid paralysis, loss of deep tendon reflexes and tenderness along the course of the peripheral nerves. Characteristic of this syndrome is an increase in the protein content without appreciable change in the cells of the spinal fluid. The etiologic agent is obscure. Most evidence, however, points to a neurotropic virus.

LESIONS ASSOCIATED WITH PATHOLOGIC DISORDERS OF THE VERTEBRAL COLUMN

Radicular pain to the shoulder and the arm may result from stimulation of the cervical and the upper thoracic nerve roots or involvement of the deep mesodermal tis-

ence of other signs of syphilis of the central nervous system. However nonspecific lesions will have a negative blood and spinal fluid serology. Excision of the posterior dural covering of the cervical spinal cord, followed by irradiation has been followed by marked relief of symptoms in some instances.

PLATYBASIA (BASILAR COMPRESSION)

Platybasia is a skeletal deformity characterized by malformation of the occipital bone and the upper cervical vertebrae. The foramen magnum is misshapen and narrow the atlas as a rule, is fused with the occipital bone, the clivus is displaced upward and the odontoid process compresses the medulla and the cervical segments of the spinal cord. Other associated anomalies are syringomyelia of the cervical segment of the spinal cord and the Klippel Feil deformity.

Gustafson and Oldberg are of the opinion that platybasia is responsible for the formation of cystic defects in the spinal cord similar to that of syringomyelia. List points out that neurologic manifestations associated with this skeletal deformity may result from mechanical compression of the brain stem or developmental defects of the central nervous system. Many etiologic factors responsible for platybasia have been cited including osteomalacia, rickets and osteitis deformans. However most investigators believe that the lesion is a developmental defect. Trauma is not considered a primary causative factor although it may influence the course of the disease.

The signs and symptoms and the character of the clinical course depend upon the degree of mechanical compression and the response of the arachnoid to irritation. Both factors are capable of causing a partial or complete block of the subarachnoid space. It is apparent that the resultant syndrome is variable and may be confused with syringomyelia, intramedullary tumor and progressive spastic paralysis. In most instances roentgenographic examination dis-

closes the true nature of the lesion. Relief of symptoms may be attained by decompression of the foramen magnum.

HERPES ZOSTER

Herpes zoster is one of the less common causes of pain in the shoulder and the arm. It is an acute infectious disease whose etiologic agent has been definitely established as a filtrable virus. The seat of involvement is the posterior root ganglia, posterior roots and posterior gray horns of the spinal cord. In more severe forms, the inflammatory process may extend into the anterior roots and give rise to motor disorders. This disease is rarely encountered in the young age groups, it occurs most often after the fourth decade of life. Implication of the cervical and the upper thoracic sensory nerve roots produces radicular pain in the neck, the shoulder or the arm which is segmental in nature and has a pattern of cutaneous distribution depending upon the specific root or roots involved.

This disease is ushered in by hyperalgesia and hypersensitiveness in a specific dermatome. The pain may be intense and of a burning character. From 3 to 5 days after the onset small discrete vesicles appear in the same segmental area corresponding to the affected root or roots. Pain is usually severe throughout the entire acute stage. A brown pigmentation of the area remains after the vesicles disappear and may persist for an indefinite period. Postherpetic intractable pain may ensue particularly in older individuals and it may persist for many years. Such distressing sequela often fails to respond to any form of therapy. In most cases section of the affected roots fails to alleviate the pain.

Although rare segmental motor involvement may occur it is a more serious problem in the upper extremity than elsewhere. The principal features are weakness or flaccid paralysis of muscles, atrophy and loss of deep reflexes. In many cases prolonged loss of motion in the scapulohumeral

contraction of the nerve root and radicular pain (Turner and Oppenheimer). Others do not agree, believing that narrowing of the intervertebral foramina in the vertical diameter (which occurs with degeneration of the intervertebral disks) does not constrict the exits of the nerves sufficiently to produce compression (Semmes and Murphy).

In spite of the controversy, it is a clinical fact that root pain in one or both upper extremities and other manifestations of nervous origin, such as numbness and tingling in the fingers, and atrophy and weakness of the muscles of the hands do exist along with osteo-arthritis of the cervical region of the vertebral column. Again it must be pointed out that irritation of such mesodermal tissues as periosteum, fascia and ligament implicated in this chronic inflammatory process, may give rise to deep segmental pain. However it must be admitted that in many instances of root pain associated with osteo-arthritis of the cervical spine the true etiologic factor is not the arthritis *per se* but protrusion of the intervertebral disks. This causative agent will be considered subsequently.

PRIMARY AND METASTATIC NEOPLASMS

Primary bone neoplasms are rarely encountered in the cervical vertebrae. Root pain in the upper limb may result from encroachment of the nerve roots by neoplastic tissue or bone destruction may cause impingement of the roots in the bony canal. Plasma-cell myeloma must be considered in patients over 40 years of age who complain of radicular pain in the shoulders and arms, who complain of neck stiffness and who on radiographic studies disclose punched-out discrete areas of bone destruction in the cervical vertebrae. Osteolytic metastatic bone lesions may exhibit a similar radiographic picture. In the latter group however a primary lesion is usually demonstrable. In the former study of the bone marrow obtained by sternal puncture confirms the diagnosis.

Metastatic bone lesions to the cervical spine are not rare. Lesions secondary to breast or thyroid cancer may produce extensive destruction of the affected vertebrae and compression of the cervical roots. Pain may be severe and intractable, more severe at night, deep and boring in character, and it may be relieved only by large doses of opiates. In two such cases a craniotomy in the cervical portion of the cord had to be performed in order to obtain relief.

TRAUMATIC LESIONS

Injuries to the cervical vertebrae producing compression of the cervical roots or the cord or both are relatively common. Root pain in the shoulders and the arms may be the most prominent subjective feature of the clinical picture. The syndrome may exist without radiographic evidence of fracture or fracture-dislocation of the cervical spine. In these instances there is reason to believe that luxation and spontaneous reduction of the spine occur at the time of the injury (whiplash mechanism). During luxation and reduction partial or complete rupture of the posterior interarticular ligaments may occur producing pain which has segmental distribution similar to that caused by nerve root pressure or irritation (Davis). In one case pain in the right deltoid region and along the dorsolateral aspect of the arm and the radial border of the forearm was the most significant symptom. Radiographic investigation disclosed no evidence of fracture or dislocation of the cervical vertebrae. Routine lateral views revealed a straight cervical spine with complete obliteration of the normal anterior curve. Lateral views with the cervical spine flexed (chin-chest position) disclosed luxation of the fourth on the fifth cervical vertebra (Fig. 775).

Davis points out that luxation of the cervical spine is a frequent cause of shoulder and arm pain that straightness in routine lateral views is indicative of some obscure pathologic disorders in the cervical region and that luxations are best treated by a hyperextension collar. He records that fall

sues by diseases affecting the cervical and the thoracic regions of the vertebral column. The most common are

- 1 Tuberculosis
- 2 Rheumatoid arthritis (ankylosing spondylitis)
- 3 Hypertrophic arthritis
- 4 Primary and metastatic neoplasms
- 5 Traumatic lesions (fracture fracture dislocations and subluxations)
- 6 Protrusion of the intervertebral disks

TUBERCULOSIS

In many cases of tuberculosis of the cervical vertebrae root pains in the neck, the shoulder and the arm are outstanding subjective findings. In one individual radiating pain in the shoulder and the arm was of such severity that it obscured all objective physical findings for many months. Pain in these cases is due both to irritation of the cervical nerve roots and to destruction of the tissues of the vertebral column.

Other characteristic features are helpful in establishing the diagnosis. Stiffness and spasm of the posterior muscles of the neck are invariably present. With destruction of the vertebral bodies a kyphosis or gibbus appears in the cervical region. All movements of the neck are guarded carefully. Roentgenographic study confirms the diagnosis by revealing destruction and collapse of the vertebral bodies forming a frank kyphotic deformity or in earlier cases narrowing of the intervertebral disk associated with some destruction of the bodies with minimal or no spinal deformity. Advanced demineralization of the affected and adjacent vertebral bodies and the presence of a soft tissue mass in the region of the osseous involvement are other radiographic characteristics of tuberculosis (Fig. 274).

Alterations in the configuration of the spinal canal and distortion of the vertebral column resulting from destruction of the osseous and ligamentous structure progress slowly. The spinal cord readily adjusts itself even to extreme deformities of the column. Manifestations pointing to compression of

the cord usually develop gradually and are produced by extension of the inflammatory process into the epidural space and compression of the cord by the expanding granulosomatous tissue.

Early signs of cord compression are spasticity, increased deep tendon reflexes, loss of abdominal reflexes and extensor plantar phenomena, all indicating that the lateral tracts are implicated first. Abnormal sensory reactions develop later below the level of the lesion. Rarely, sudden collapse of the vertebral bodies may occur, producing acute compression of the cord manifested by a flaccid paralysis. Cervical nerve roots may also be compressed by the tuberculomatous mass giving rise to segmental pain. More than one root is usually involved, confusing the segmental pattern of distribution. The cervical segments of the spinal column are less frequently involved than the dorsal and the lumbar regions.

RHEUMATOID ARTHRITIS

Segmental root pain in the neck and the upper limb is commonly found in the early stages of ankylosing spondylitis involving the cervical vertebrae. In these instances, pain is the result of constriction and irritation of the nerve roots by the inflammatory process implicating the tissues which form the intervertebral foramina. It is doubtful whether any actual bony impingement of the nerve roots exists in these instances. Pain disappears when the acute inflammatory process subsides and bony ankylosis of the vertebral column ensues.

HYPERTROPHIC ARTHRITIS

Varying degrees of segmental pain in intensity are frequently a concomitant clinical feature of hypertrophic arthritis in the cervical spine. Many workers are of the opinion that osteo-arthritis of this region results from thinning of the intervertebral disks and plays no part in the production of the syndrome. They believe that thinning of the disks is responsible for narrowing of the intervertebral foramina thereby causing

ure to treat adequately the early cases to result in creeping migration of one vertebral body on the one below which is responsible for segmental pain in the upper extremity. Old lesions may also be treated by a hyperextension collar if the syndrome recurs. Davis recommends spinal fusion. Luxations may be complicated by protrusion of an intervertebral disk which causes pain by direct irritation of the corresponding cervical nerve root.

PROTRUSION OF THE INTERVERTEBRAL DISK

Since the report of Semmes and Murphey, in 1943, emphasized the frequency of extrusion of the intervertebral disks in the cervical region as causative agents for segmental pain in the shoulder and the arm and for sensory disturbances in the finger, it has become apparent that many of these cases had paralleled hitherto under such erroneous diagnoses as brachial neuritis, cervical rib or scalenus anticus syndrome and arthritis of the cervical spine.

Although this is true, many other factors may produce a similar clinical picture. For example, the numerous anatomic aberrations of the thoracic operculum may be the basis for mechanical brachial neuritis. When the etiologic agent is uncertain, it becomes apparent that segmental pain in the upper extremity demands careful scrutiny and study before a diagnosis is established. Failure to appreciate the significance of this statement will result in mismanagement of these cases. It is common knowledge that many individuals are victims of needless surgery because of failure to search for and evaluate properly all the numerous possibilities that may be responsible for this distressing symptom complex.

Increasing knowledge of this entity stresses that the symptomatology is variable depending upon the location and the size of the lesion. Prior to the study of Semmes and Murphey (1943) only large protrusions of the disks were recognized. These produced unilateral or bilateral pressure on the cord and simulated extramedullary neoplasms or other neurologic diseases.

Small lateral herniations of the disks causing root pain were considered to be extremely rare.

Clinical investigation has reversed this view. Small lateral extrusions of the disks are now considered to be relatively common and are the causative factor in the greater majority of patients exhibiting this syndrome. On the other hand, large central herniations of the disks causing compression of the cord are rarely encountered.

Lateral protrusions are observed more frequently in men than in women and they occur more frequently on the left than on the right side. The cervical nerve roots which are implicated most frequently are in order of frequency the seventh, the sixth and the eighth.

There appears to be no correlation between severe trauma to the cervical spine and the radicular syndrome of herniation of intervertebral disks. More often the syndrome is initiated by such minor injuries as sudden, unguarded movements of the head. In some instances recurrent episodes of stiff neck or cricks in the neck antedate the onset of the complaints by many months or even years. As pointed out by Semmes and Murphey, stiffness and cricks in the neck may not be the result of fibrosities or some focus of infection, but the result of stretching and tearing the annulus fibrosis of a degenerating cervical disk which are precursors of disk extrusion.

The onset of the disease is generally initiated by a feeling of soreness and stiffness in the neck, then pain of varying intensity radiating to the shoulder, the upper arm, the forearm, and occasionally to the hands and the fingers, becomes the dominant symptom. Paresthesias and sensations of numbness are the outstanding symptoms observed in the hand and the fingers. Rarely there may be paresthesias without pain or pain without paresthesias. The pain usually is sharp and lancinating and follows a segmental pattern. It is accentuated or reproduced by certain movements of the neck and by acts which increase intra abdominal and intrathoracic pressure (Eaton).

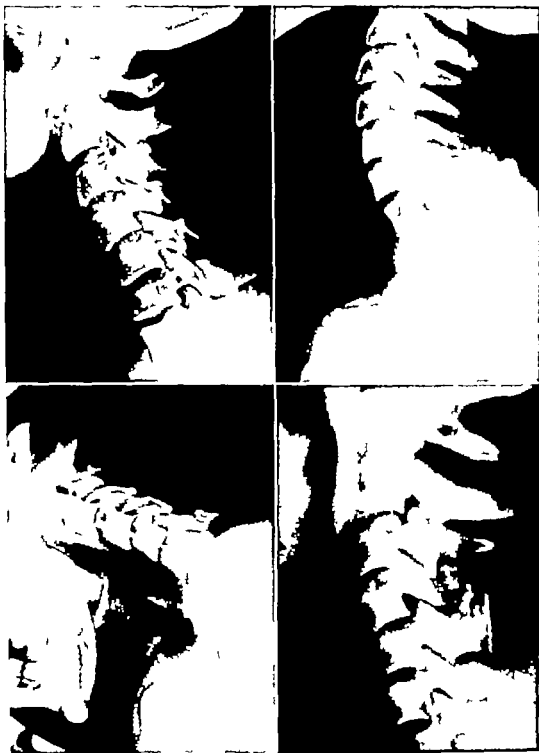


FIG 275 (*Top left*) Lateral view of cervical spine of male, 21 years old 8 weeks following trauma to the cervical spine (by whip-lash mechanism) Observe that the normal forward cervical curve is straightened particularly in the segment formed by the fourth, fifth and sixth cervical vertebrae. (*Top right*) Lateral view with spine hyperextended (same patient) (*Bottom left*) Lateral view with spine hyperflexed (same patient) Note the forward displacement of the fourth cervical on the fifth cervical vertebra. The outstanding symptom in this case was pain radiating into both shoulder and right arm accentuated by forward bending of the neck. Observe the increased interval between the spinous processes of the fourth and fifth vertebrae. (*Bottom right*) Same patient 7 months after injury Note the alteration of the cervical curve and increased forward displacement of the fourth on the fifth cervical vertebra ("creeping migration") This roentgenogram was taken 8 weeks after a fusion of the fourth and fifth cervical vertebrae had been done. Coalescence of the bone chips is discernible. This patient was seen for the first time 8 weeks after the injury (his head was snapped forward when the automobile he was driving was forcibly struck in the rear) No treatment had been rendered during this period. A hyperextension collar was then applied which relieved the symptoms he wore the collar for 12 weeks symptoms returned when the collar was removed A spinal fusion (as shown above) was then performed

ure to treat adequately the early cases results in creeping migration of one vertebral body on the one below, which is responsible for segmental pain in the upper extremity. Old lesions may also be treated by a hyperextension collar, if the syndrome recurs. Davis recommends spinal fusion. Luxations may be complicated by protrusion of an intervertebral disk which causes pain by direct irritation of the corresponding cervical nerve root.

PROTRUSION OF THE INTERVERTEBRAL DISK

Since the report of Semmes and Murphey in 1943 emphasized the frequency of extrusion of the intervertebral disks in the cervical region as causative agents for segmental pain in the shoulder and the arm and for sensory disturbances in the fingers, it has become apparent that many of these cases had paraded hitherto under such erroneous diagnoses as brachial neuritis, cervical rib or calenus anticus syndrome and arthritis of the cervical spine.

Although this is true many other factors may produce a similar clinical picture. For example the numerous anatomic aberrations of the thoracic operculum may be the basis for mechanical brachial neuritis. When the etiologic agent is uncertain it becomes apparent that segmental pain in the upper extremity demands careful scrutiny and study before a diagnosis is established. Failure to appreciate the significance of this statement will result in mismanagement of these cases. It is common knowledge that many individuals are victims of needless surgery because of failure to search for and evaluate properly all the numerous possibilities that may be responsible for this distressing symptom complex.

Increasing knowledge of this entity stresses that the symptomatology is variable depending upon the location and the size of the lesion. Prior to the study of Semmes and Murphey (1943) only large protrusions of the disks were recognized. These produced unilateral or bilateral pressure on the cord and simulated extramedullary neoplasms or other neurologic diseases.

Small lateral herniations of the disks causing root pain were considered to be extremely rare.

Clinical investigation has reversed this view. Small lateral extrusions of the disks are now considered to be relatively common and are the causative factor in the greater majority of patients exhibiting this syndrome. On the other hand large central herniations of the disks causing compression of the cord are rarely encountered.

Lateral protrusions are observed more frequently in men than in women and they occur more frequently on the left than on the right side. The cervical nerve roots which are implicated most frequently are in order of frequency the seventh, the sixth and the eighth.

There appears to be no correlation between severe trauma to the cervical spine and the radicular syndrome of herniation of intervertebral disks. More often the syndrome is initiated by such minor injuries as sudden, unguarded movements of the head. In some instances recurrent episodes of stiff neck or cricks in the neck antedate the onset of the complaints by many months or even years. As pointed out by Semmes and Murphey stiffness and cricks in the neck may not be the result of fibrositis or some focus of infection, but the result of stretching and tearing the annulus fibrosis of a degenerating cervical disk which are precursors of disk extrusion.

The onset of the disease is generally initiated by a feeling of soreness and stiffness in the neck, then pain of varying intensity radiating to the shoulder, the upper arm, the forearm and occasionally to the hands and the fingers becomes the dominant symptom. Paresthesias and sensations of numbness are the outstanding symptoms observed in the hand and the fingers. Rarely there may be paresthesias without pain or pain without paresthesias. The pain usually is sharp and lancinating and follows a segmental pattern. It is accentuated or reproduced by certain movements of the neck and by acts which increase intra abdominal and intrathoracic pressure (Eaton).

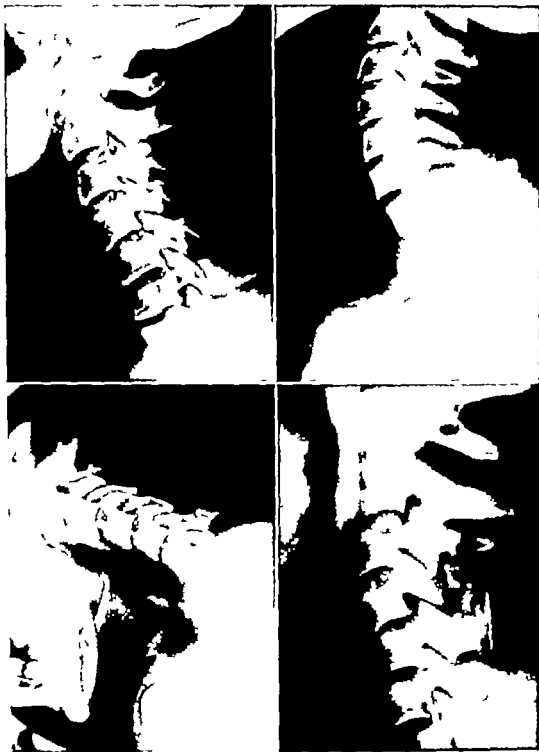


FIG 275 (Top left) Lateral view of cervical spine of male 21 years old 8 weeks following trauma to the cervical spine (bv whip-lash mechanism) Observe that the normal forward cervical curve is straightened particularly in the segment formed by the fourth, fifth and sixth cervical vertebrae. (Top right) Lateral view with spine hyperextended (same patient) (Bottom left) Lateral view with spine hyperflexed (same patient) Note the forward displacement of the fourth cervical on the fifth cervical vertebra. The outstanding symptom in this case was pain radiating into both shoulder and right arm accentuated by forward bending of the neck. Observe the increased interval between the spinous processes of the fourth and fifth vertebrae. (Bottom right) Same patient 7 months after injury Note the alteration of the cervical curve and increased forward displacement of the fourth on the fifth cervical vertebra ("creeping migration") This roentgenogram was taken 8 weeks after a fusion of the fourth and fifth cervical vertebrae had been done. Coalescence of the bone chips is discernible. This patient was seen for the first time 8 weeks after the injury (his head was snapped forward when the automobile he was driving was forcibly struck in the rear) No treatment had been rendered during this period. A hyperextension collar was then applied which relieved the symptoms he wore the collar for 12 weeks symptoms returned when the collar was removed. A spinal fusion (as shown above) was then performed.



FIG 277 (Left) Unusually large transverse processes of the seventh cervical vertebrae
 FIG 278 (Right) Aberration of the thoracic outlet (bilateral) associated with large cervical ribs and abnormal first thoracic ribs, and an elevated scapula on the left side

Involvement of the anterior root fibers result in varying degrees of atrophy, weakness and fibrillations in the muscles innervated by the affected root. Response of deep tendon reflexes of the biceps brachii and the triceps muscles may be altered. Diminished or absent tendon reflex of the biceps brachii indicates encroachment of the sixth cervical nerve root. Diminished or absent tendon reflex of the triceps points to implication of the seventh cervical nerve root. Weakness and atrophy of the muscles of the hand are consistent with involvement of the eighth cervical nerve root and atrophy and weakness of the deltoid with involvement of the sixth nerve root.

Many established entities, which may produce radicular pain must be considered in the differential diagnosis. Important among these are such aberrations of the thoracic outlet as cervical rib and abnormal first thoracic rib, scalenus anticus syndrome, neoplasms of the spinal cord and lesions affecting the shoulder region, such as bicipital tenosynovitis, calcareous tendinitis, lesions of the musculotendinous cuff and frozen shoulder. All these must receive due consideration and they must be excluded by careful physical examination and radiographic studies before diagnosis of protrusion of an intervertebral disk is made.

Valuable information is obtained by ade-

quate radiographic examination. The most significant observation is straightness of the spinal column in the cervical region. Lateral views may reveal thinning of the involved intervertebral disk and tipping of the anterior and the posterior margins of the adjacent vertebrae (Fig 276).

Myelography, using pantopaque, localizes the lesion in a high percentage of cases and is considered a valuable diagnostic procedure. Oblique and stereoscopic views will bring into view the intervertebral foramina and disclose any alteration in their configuration and the presence of bony spurs encroaching on the foramina.

Conservative management should be given a trial before surgical intervention is considered. Many cases of mild or moderate severity are relieved by restricting movement of the neck by a collar. Physiotherapy in the form of radiant heat is a valuable adjunct. Traction made with a head halter will relieve many. Manipulation is contraindicated at all times.

Failure to respond to conservative measures warrants surgical intervention which comprises exploration of the affected nerve root and removal of the extruded disk. In cases with large protrusions causing spinal cord compression, surgical intervention certainly should be the primary treatment of choice.

Maneuvers such as the Lasègue maneuver, extreme flexion of the cervical spine and downward traction on the arm which stretches the affected nerve roots also intensify the pain (Eaton)



FIG 276 Lateral view of a female aged 43. Observe the straightness of the cervical spine and thinning of the intervertebral disk between the fifth and sixth cervical vertebrae whose adjacent anterior and posterior margins reveal advanced lippling. Conservative measures failed to alleviate the symptoms. An extruded disk was found at operation on the left side; its removal resulted in disappearance of all symptoms.

Pain may be more pronounced during rest when the body assumes a horizontal position. This position results in elongation of the spinal column, thereby stretching the implicated nerve roots (Eaton). Assuming the upright position alleviates the pain. Spurling's "foraminal compression test," in which pressure is applied to the top of the head while the head and the neck are flexed laterally toward the affected side, reproduces the radicular pain in most cases espe-

cially during the acute phases of the disease. Flexion of the neck laterally toward the unaffected side alleviates the pain in some cases but fails in others. Areas of tenderness may be demonstrable in the shoulder, the arm and the wrist. Pressure over the point of exit of the affected nerve root from its intervertebral foramen elicits marked tenderness (Semmes and Murphy).

The segmental pattern of pain and other sensory manifestations will depend upon the specific nerve root implicated, a feature of considerable aid in localizing the lesion. Compression of the sixth nerve root produces pain in the shoulder along the dorso-lateral aspect of the arm, outer (radial) surface of the forearm, and occasionally in the thumb and the index finger. Paresthesias and sensations of numbness are felt chiefly in the thumb and at times in the index finger. Implication of the seventh root discloses the same pain distribution as the sixth nerve root. However, as recorded by Semmes and Murphy, sensory phenomena are concentrated chiefly in the index finger. These authors also point out that pain radiating to the precordial region is often a concomitant feature of seventh cervical root involvement and may be confused with angina. Compression of the eighth cervical root gives rise to sensory phenomena along the inner (ulnar) aspect of the arm, the forearm and the fifth finger; often the fourth finger is affected. Pain is also referred to the shoulder region.

Motor phenomena are exceedingly variable and depend upon the level, the size, the location and the duration of the lesion. Large extrusions approaching the midline may produce a syndrome similar to an extradural neoplasm compressing the cord and they may cause sensory and motor disturbances below the level of the lesion; these are rare lesions. More commonly the protrusion occupies a lateral position causing pressure on the dorsal sensory nerve root. Lesions of long duration may also implicate some fibers of the anterior motor nerve root.

the transverse processes to complete ribs which join with the first thoracic ribs anteriorly

Between these two types numerous variations are encountered, some of which join the first thoracic ribs. Most often they occur unilaterally but they may occur bilaterally. In cases with bilateral ribs one side may vary considerably in length and configuration from the other. Usually, those with which one is chiefly concerned clinically arise from the seventh cervical but they may arise from any of the cervical vertebrae. At times it may be difficult to distinguish an abnormal first thoracic from a cervical rib. Indeed it is doubtful whether cervical ribs per se are responsible for symptoms. Evidence seems to indicate that other factors must be associated with this anomaly before symptoms arise. Chief among these factors are

- 1 Abnormal position of the shoulder in relation to the thorax due to congenital or developmental aberrations

- 2 The presence of a postfixed plexus whose lower fibers must run upward sharply and obliquely to cross the cervical rib in order to reach the lower trunk of the plexus. As their course is directed downward into the arm these fibers become acutely angulated over the rib. The same abnormal topographic anatomy is observed in the absence of a cervical rib and in the presence of a high first thoracic rib associated with a postfixed plexus.

- 3 Descent of the shoulder resulting from loss of normal muscle tone, occupations which tend to depress the shoulders and trauma.

Walshe, Jackson and Wyburn-Mason postulate that anatomic and developmental factors are responsible for numerous anomalies of the thoracic operculum which often result in narrowing of the interval between the clavicle and the upper thoracic cage. The clavicle in these instances during arm motion compresses the neurovascular structures against the abnormal costal elements. In cases lacking these elements, drooping

of the shoulder may result in diminution of the costoclavicular space rendering the brachial plexus and the subclavian vessels vulnerable to compression by the clavicle. Walshe, Jackson and Wyburn-Mason also contend that repeated minor trauma to soft tissue maladjusted to the thoracic outlet and clavicle may produce during arm motion secondary inflammatory changes around the nerve trunks and the subclavian vessels, which in turn may give rise to phenomena of compression.

Alson expressed the belief that the scalenus anticus muscle was the prime etiologic factor of the syndrome regardless of whether a cervical rib was present or not. He contends that during the period of greatest muscular development the individual indulges in strenuous physical activities which tend to cause drooping of the shoulders and that during this period of life the muscle is more apt to compress the neurovascular structures than at other periods. Vassfager and Grant support Alson's theory and add that removal of a portion of the cervical rib in addition to scalenotomy is sometimes necessary to alleviate the symptoms.

Women are affected more frequently than men. Despite the fact that in many instances bilateral cervical ribs exist the right side exhibits such manifestations more often than the left. The cardinal features of the syndrome are pain in the region of the shoulder, usually radiating to the elbow and the ulnar aspect of the forearm, the hand and the fourth and the fifth fingers. Parasthesias, such as tingling, formication and numbness in the area of pain distribution in the forearm and the hand, are concomitant subjective symptoms. Although pain is projected most often to the region of the deltoid muscle, occasionally it is referred to the scapular, the pectoral and the occipital regions and along the outer aspect of the forearm. In general the sensory phenomena point to involvement of the lower two nerve trunks but at times all three trunks may be implicated.



FIG 279 (*Left*) Lateral view of the cervical spine of a case of Kippel Feil deformity. There is fusion of the cervical bodies and their posterior elements. (*Right*) Anteroposterior view of a case of Kippel Feil deformity. Observe the pronounced asymmetry of the thoracic outlet and irregular formation of the lower cervical and upper thoracic vertebrae. On the right there is also a Sprengel's scapula.

LESIONS SITUATED OUTSIDE THE VERTEBRAL COLUMN

Numerous mechanical factors in the supraclavicular region are capable of producing compression of the brachial plexus. Lack of knowledge and failure to comprehend the mechanism of these variable factors is often responsible for erroneous diagnoses. From a clinical viewpoint these entities are apt to produce similar signs and symptoms especially in the early stages of the disease. Thus it becomes apparent that careful clinical analysis is essential and all helpful diagnostic aids should be utilized to establish a correct diagnosis and institute proper treatment.

The more common lesions producing syndromes of mechanical compression of the brachial plexus are

- 1 Hypertrophied or spastic scalenus anticus muscle
- 2 Anomalies of the scalene muscles
- 3 Anomalies of the thoracic outlet such as cervical rib, abnormal first thoracic rib, cervicodorsal scoliosis and Keppel Feil deformity
- 4 Aberrations of the brachial plexus
- 5 Compression of the subclavian vessels and the brachial plexus between the clavicle and the first thoracic rib

- 6 Compression of the subclavian vessels and the brachial plexus by hyperabduction of the arm (subcoracoid pectoralis minor syndrome)

CERVICAL RIB SYNDROME

Numerous anomalies, both of the costal elements and the brachial plexus, occur in the cervicothoracic region. Generally, they occur in combination. Aberrations of the thoracic outlet are commonly associated with anomalies of the brachial plexus. For example a rudimentary first thoracic rib and a postfixed plexus always seem to be associated. The presence of a cervical rib does not produce symptoms of compression of the brachial plexus and the subclavian vessels in all instances. In fact as will be seen the syndrome may exist without cervical anomalies or first thoracic rib.

Cervical ribs are uncommon. Eaton records that a survey of 7 706 roentgenograms of the thorax disclosed 45 cases of one or more cervical ribs, 29 of which were in women. Roughly 50 per cent of the cases with cervical ribs exhibit manifestations of nervous or vascular compression. Aberrant cervical ribs exhibit considerable variation in length, shape and direction. They may form small processes projecting just beyond

the transverse processes to complete ribs which join with the first thoracic ribs anteriorly.

Between these two types numerous variations are encountered some of which join the first thoracic ribs. Most often they occur unilaterally, but they may occur bilaterally. In cases with bilateral ribs one side may vary considerably in length and configuration from the other. Usually those with which one is chiefly concerned clinically arise from the seventh cervical but they may arise from any of the cervical vertebrae. At times it may be difficult to distinguish an abnormal first thoracic from a cervical rib. Indeed it is doubtful whether cervical ribs per se are responsible for symptoms. Evidence seems to indicate that other factors must be associated with this anomaly before symptoms arise. Chief among these factors are:

1. Abnormal position of the shoulder in relation to the thorax due to congenital or developmental aberrations.

2. The presence of a postfixed plexus whose lower fibers must run upward sharply and obliquely to cross the cervical rib in order to reach the lower trunk of the plexus. As their course is directed downward into the arm these fibers become acutely angulated over the rib. The same abnormal topographic anatomy is observed in the absence of a cervical rib and in the presence of a high first thoracic rib associated with a postfixed plexus.

3. Descent of the shoulder resulting from loss of normal muscle tone, occupations which tend to depress the shoulders and trauma.

Walshe, Jackson and Wyburn-Mason postulate that anatomic and developmental factors are responsible for numerous anomalies of the thoracic operculum which often result in narrowing of the interval between the clavicle and the upper thoracic cage. The clavicle in these instances during arm motion compresses the neurovascular structures against the abnormal costal elements. In cases lacking these elements drooping

of the shoulder may result in diminution of the costoclavicular space rendering the brachial plexus and the subclavian vessels vulnerable to compression by the clavicle. Walshe, Jackson and Wyburn-Mason also contend that repeated minor trauma to soft tissue maladjusted to the thoracic outlet and clavicle "may produce during arm motion secondary inflammatory changes around the nerve trunks and the subclavian vessels which in turn may give rise to the phenomena of compression."

Adson expressed the belief that the scalenus anticus muscle was the prime etiologic factor of the syndrome regardless of whether a cervical rib was present or not. He contends that during the period of greatest muscular development the individual indulges in strenuous physical activities which tend to cause drooping of the shoulders and that during this period of life the muscle is more apt to compress the neurovascular structures than at other periods. Naffziger and Grant support Adson's theory and add that removal of a portion of the cervical rib in addition to scalenotomy is sometimes necessary to alleviate the symptoms.

Women are affected more frequently than men. Despite the fact that in many instances bilateral cervical ribs exist the right side exhibits such manifestations more often than the left. The cardinal features of the syndrome are pain in the region of the shoulder usually radiating to the elbow and the ulnar aspect of the forearm, the hand and the fourth and the fifth fingers. Paresthesias such as tingling, formication and numbness in the area of pain distribution in the forearm and the hand are concomitant subjective symptoms. Although pain is projected most often to the region of the deltoid muscle occasionally it is referred to the scapular, the pectoral and the occipital regions and along the outer aspect of the forearm. In general, the sensory phenomena point to involvement of the lower two nerve trunks but at times all three trunks may be implicated.

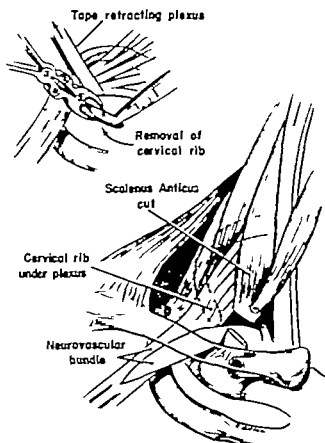


FIG. 280. Operative procedure for relief of scalenus anticus syndrome with cervical rib (*Bottom*) Anatomic relationship of the structures producing the syndrome division of scalenus anticus may not suffice to relieve tension of the nerve trunks and constriction of the subclavian artery (*Top*) Resection of a portion or all of the rib may be necessary

The character of the pain is variable. It may be deep boring and continuous or sharp and lancinating. It may be reproduced or accentuated by acts which depress the shoulder or may be relieved by elevating the shoulder. Many of these patients sleep with the arm above the head. Tilting the head laterally toward the affected side also relieves the pain, since this position takes all tension off the scalene muscles and the brachial plexus. In the light of the work of Wright who showed conclusively that of 150 normal individuals he was able to obliterate the radial pulse in 125 on the right side and on the left side in 124 by hyperabduction of the arm, it is difficult to

explain why elevation of the arm relieves pain.

Such abnormal motor phenomena as muscular atrophy, weakness and fasciculations manifest themselves in the later stages of the disease. These changes in the interossei, the hypothenar muscles and the adductor of the thumb point to implication of the ulnar nerve. Changes in the hyperthenar muscles (abductor pollicis brevis and apponens pollicis) suggest implication of the median nerve (Wilson). Motor manifestations involving the ulnar nerve are encountered more frequently particularly in the presence of a cervical rib.

Vascular phenomena are observed sometimes in addition to manifestations of nervous origin. These consist of swelling, coldness, tingling and cyanosis of the hand and the fingers. Cases are on record of gangrene of the tips of the fingers. In extreme cases the vascular symptoms may be confused with Raynaud's disease or thrombo-angitis obliterans.

Considerable controversy exists as to the factors responsible for vascular phenomena. Some authorities believe that they are the result of varying degrees of occlusion of the subclavian vessels. An arterial bruit resulting from compression of the subclavian artery and with or without a thrill may be heard in the supraclavicular region. Walshe Jackson and Wyburn emphasize that vascular symptoms and signs are caused by compression of the subclavian artery against the costal elements by the clavicle. They describe three syndromes of vascular origin.

- 1 The relatively common syndrome of recurrent coldness, cyanosis, pallor and tingling of the hand and the digits associated with transient obliteration of an otherwise normal radial pulse by placing the arm in certain positions.

- 2 The syndrome of patent aneurysmal dilatation of the third part of the subclavian artery. This may include the components already named and increased pulsation and bruit in addition, with or without a thrill over the subclavian artery above the clavicle.

- 3 The syndrome of a completely or partly occluded aneurysmal dilatation of the third



FIG. 281 (*Left*) Bilateral cervical ribs in a female aged 22. Pain was projected to the right shoulder, elbow, and ulnar surface of the forearm and into the fourth and fifth fingers. Atrophy of the muscles of the hypothenar eminence existed. No symptoms were present in the left extremity. (*Right*) Anteroposterior view of the patient at left. After division of the scalenus anticus muscle it was noted that the nerve trunks were tightly stretched over the anterior portion of the rib. This segment of the rib was resected.

part of the subclavian artery, consisting of signs of ischemia in the limb and sometimes of embolism in the digits leading to small regions of gangrene.¹

Other investigators support the hypothesis that vascular symptoms follow pressure on the sympathetic fibers in the first thoracic nerve root at the point where it is angulated and stretched over the rib (Stopford and Telford Wilson). Cases of Horner's syndrome associated with cervical rib syndrome have been reported. In some instances it is evidence that the sympathetic trunks are implicated although it must be admitted that the seat of involvement may be in the stellate ganglion.

SCALENUS ANTICUS SYNDROME

It is general knowledge that the symptom complex just described is encountered also in patients with no cervical ribs. In fact a cervical rib is demonstrable radiographically in the minority of the patients who exhibit the syndrome. Since the report of

Alson and Coffey (1927) (in which is described relief of nervous and vascular pressure symptoms by division of the scalenus anticus muscle alone in cases with and without cervical ribs) the role of this muscle has assumed considerable prominence. Ochsner, Gage and DeBakey (1935) observed at operation that the scalenus muscle which is innervated by fibers from the brachial plexuses, was hypertrophied and spastic; they attributed its hypertonicity and increased size to irritation of the brachial plexus by a normal first thoracic rib. The observers contend that a low shoulder girdle, high sternum, high first thoracic rib or a postfixed brachial plexus alone or in combination may be responsible for irritation of the plexus by a normal first rib. Irritation of the plexus in turn causes spasm and hypertrophy of the scalenus muscle. The spastic muscle forces the first rib upward causing further irritation of the brachial plexus, thereby establishing a vicious cycle.

It is apparent that any condition capable of causing sagging of the shoulders may institute the syndrome. Such conditions are muscular atonia observed in debilitating

¹Walsh, F. M. R., Jackson, H., Wyburn, M. R. On some pressure effects associated with cervical and rudimentary "normal" first ribs, *Brain* 67: 164, 1944.

diseases nervous disorders, occupations which tend to depress the shoulders, trauma, producing a forceful downward thrust on the shoulders and fatigue. This is especially true if the afore-mentioned anatomic and developmental abnormalities exist. Stopford and Telford emphasize that the combination of a postfixed brachial plexus and a low shoulder girdle observed in adults in whom the lower components of the brachial plexus are stretched tightly over the normal first rib renders the individuals vulnerable to the disorder. Factors which induce further

descent of the shoulder, as previously noted, are apt to produce symptoms of compression.

Many investigators will not recognize the scalenus syndrome as a distinct entity; others maintain that the scalenus anticus muscle is only rarely the prime operative cause of irritations of the plexus. Nevertheless, numerous workers report brilliant results attained by scalenotomy alone in cases with or without cervical ribs. Critical analyses of many cases in which this procedure was carried out reveal that the results have

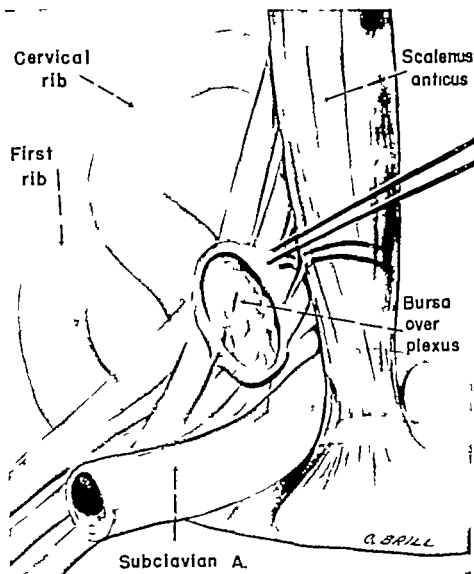


FIG 282 Drawing of an anatomic specimen showing compression of the subclavian artery and brachial plexus between the scalenus anticus muscle and a well formed cervical rib. A bursa with thickened walls had formed immediately above the nerve trunks. In the drawing, the top of the bursa has been removed to show the floor of the structure resting on the brachial plexus.

FIG 283 Brachial plexus is constricted by an anomalous formation of the scalenus anticus and scalenus medius muscles. (Allen E. V., Barker, H. W., and Hines, E. H. Jr *Peripheral Vascular Diseases Philadelphia Saunders, p 306*)

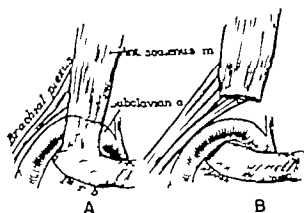
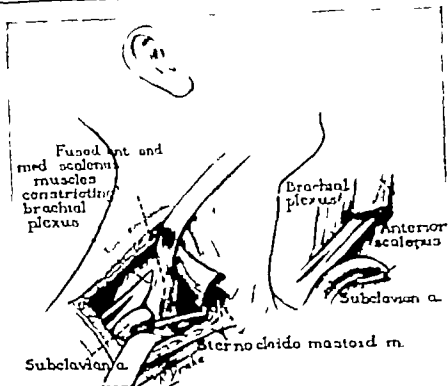


FIG 284 Relationship of structures without a cervical rib the scalenus muscle may be hypertrophied constricting the neurovascular structures against the first thoracic rib (Craig W. McK. and Knepper P. A. *Cervical rib and the scalenus anticus syndrome Ann Surg 105 556-563*)

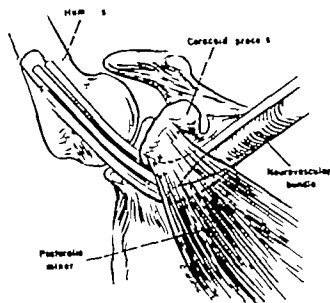


FIG 285 Drawing from anatomic specimen showing mechanism of sub-coracoid pectoralis minor syndrome. With the arm hyperabducted the neurovascular structures are pulled tightly around and beneath the coracoid process and compressed by the pectoralis minor

been good in some and poor in others, leading one to conclude that the scalenus muscle may be the prime operative factor in some instances. In others contraction and spasm of the muscle may be secondary to some other primary lesion.

A scalenus anticus syndrome may be associated with extrusions of intervertebral disks of the cervical spine, injuries to the vertebrae and ligamentous tissues in the

cervical region, compression mechanisms, rheumatoid and hypertrophic arthritis of the cervical spine, and a host of other lesions affecting this region. The syndrome may be associated with an anomalous relationship between the plexus and the scalene muscles.

diseases, nervous disorders occupations which tend to depress the shoulders, trauma, producing a forceful downward thrust on the shoulders and fatigue This is especially true if the afore-mentioned anatomic and developmental abnormalities exist Stopford and Telford emphasize that the combination of a postfixed brachial plexus and a low shoulder girdle, observed in adults in whom the lower components of the brachial plexus are stretched tightly over the normal first rib, renders the individuals vulnerable to the disorder Factors which induce further

descent of the shoulder, as previously noted, are apt to produce symptoms of compression

Many investigators will not recognize the scalenus syndrome as a distinct entity others maintain that the scalenus anticus muscle is only rarely the prime operative cause of irritations of the plexus. Nevertheless, numerous workers report brilliant results attained by scalenotomy alone in cases with or without cervical ribs. Critical analyses of many cases in which this procedure was carried out reveal that the results have

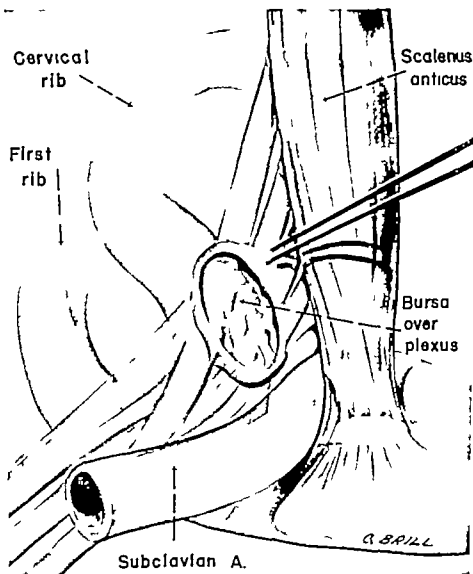


FIG 282 Drawing of an anatomic specimen showing compression of the subclavian artery and brachial plexus between the scalenus anticus muscle and a well formed cervical rib A bursa with thickened walls had formed immediately above the nerve trunks. In the drawing the top of the bursa has been removed to show the floor of the structure resting on the brachial plexus

fibers (Fig 283) The brachial plexus and the subclavian artery may be compressed between the distal ends of the scalenus anticus and the scalenus medius muscles close to their insertions into the first rib (Fig 284)

Symptoms and signs of the scalenus syndrome are similar to those of cervical rib syndrome Horner's syndrome has been observed in some cases of both syndromes Palpation of the supraclavicular fossa will reveal a firm bony prominence if a cervical rib exists Pressure over the scalenus anticus muscle elicits extreme local tenderness and aggravation of the pain along the ulnar aspect of the arm and the forearm This is true whether or not a cervical rib is present Often, on palpation, the scalenus anticus muscle on the affected side feels tense firmer and hypertrophied, as compared with the opposite side Changes in the character of the pulse and blood pressure during various maneuvers are no longer of any significant clinical value, because as will be shown subsequently, they occur in normal individuals and they are produced by other mechanisms which compress the subclavian vessels. Roentgenographic studies are essential for determining the configuration of the thoracic outlet as well as the presence of cervical or abnormal first thoracic ribs

SUBCORACOID PECTORALIS MINOR SYNDROME AND COSTOCLAVICULAR SYNDROME

Wright demonstrated that compression of the brachial plexus and the subclavian vessels occurred when the arms of normal subjects were hyperabducted This maneuver produced a neurovascular syndrome which resembled closely cervical rib and scalenus anticus syndrome Hyperabduction of the right arm above the head resulted in obliteration of the pulse in 125 of 150 normal individuals the same maneuver on the left side produced obliteration of the pulse in 124 of 150 normal individuals The level of abduction just below the level which would produce obliteration of the pulse was designated the "marginal position"

Three individuals with the arm in the marginal position could obliterate the pulse by deep respiration while in two others the pulse became more prominent Eight subjects with the arm in the marginal position could obliterate the pulse by muscular contractions He further noted that turning the head toward or away from the arm tested may open or close the subclavian artery that flexion of the cervical spine reduced the incidence of obliteration, and hyperextension increases the incidence

Investigation on anatomic specimens disclosed two mechanisms which caused compression of the neurovascular structures in no way related to a cervical rib or the scalenus anticus muscle

1 By hyperabduction of the arm the brachial plexus and the subclavian vessels are pulled tightly around and beneath the coracoid process and at the same time, compressed by the pectoralis minor muscle, the greatest amount of torsion occurring at the point of transition between the subclavian and the axillary artery and vein.

2 Compression of the subclavian vessels and the brachial plexus is effected by reduction of the costoclavicular space

Certain movements of the shoulder girdle such as pulling the shoulders backward and downward and by hyperextension of the neck compress the neurovascular structures between the posterior surface of the clavicle and the anterior surface of the first rib This mechanism was also described by Falconer and Weddell who noted that symptoms of vascular and nervous compression was a common phenomenon They were able to produce it in 25 of 50 normal men and in 30 of 50 normal women These observers explored the supraclavicular region of one patient who manifested symptoms and signs of a scalenus anticus syndrome and noted that section of the scalenus anticus muscle did not prevent obliteration of the pulse when the neck was hyperextended and the shoulders were forced downward and backward They noted at operation that the afore mentioned movements caused diminu-

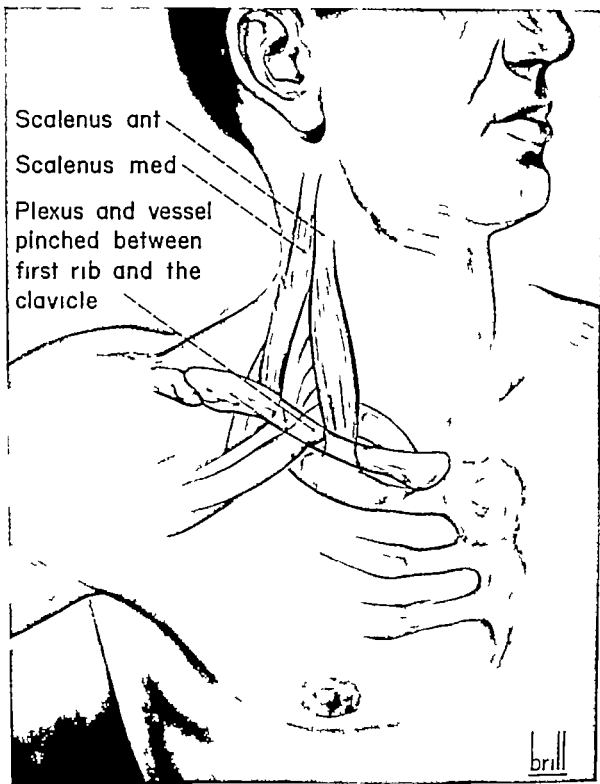


FIG 286 Drawing from anatomic specimen showing mechanism of costoclavicular syndrome. Observe that, with the shoulder drawn downward and backward, the neurovascular structures are compressed between the clavicle and first thoracic rib

Occasionally the bellies of the scalenus medius and the scalenus anticus are fused into one mass, through which traverse the trunks of the brachial plexus again the trunks may split the fibers of the scalenus

anticus muscle instead of passing through the interval between the scalenus anticus and the scalenus medius muscles. In both instances the nerve trunks may be readily compressed by the surrounding muscle

of the lung with evidence of erosion of the upper ribs, the transverse processes and in some instances, the vertebral bodies (Fig 287)

Some investigators maintain that the sympathetic manifestations (Horner's syndrome) are the result of pressure on the sympathetic fibers in the common trunk from the eighth cervical and the first thoracic. Others contend that they result from involvement of the stellate ganglion, which lies just anterior to the head and the neck of the first rib, or slightly below this level. Sensory and motor manifestations pointing to involvement of the eighth cervical and the first and the second thoracic nerve roots and Horner's syndrome are the principal features of this symptom complex.

Neoplasms arising from the sheath cells of the nerves comprising the brachial plexus anywhere along their course may be responsible for pain projected to the shoulder or the arm.

Neurinoma commonly arise from the cervical and the thoracic roots of the spinal nerves. They produce segmental sensory and motor phenomena and exhibit such characteristic features of root involvement as exacerbation of pain by increased intra thoracic or intra abdominal pressure and by maneuvers designed to stretch the nerve roots. As a rule neurinomas appear as single lesions. However, they may be multiple particularly if associated with generalized neurofibromatosis. Single neurofibromas occasionally arise from the nerves of the brachial plexus and may be demonstrable by palpation of the cervical the supraclavicular or the axillary regions. The brachial plexus may be implicated in generalized neurofibromatosis.

INFLAMMATORY LESIONS

Increasing knowledge of the numerous factors that may account for neck, shoulder and arm pain has reduced considerably the number of heterogenous entities hitherto grouped under the all inclusive term "brachial plexus neuritis."



FIG 287 Roentgenogram of the thoracic outlet of a case of Pancoast tumor. Observe the soft tissue mass in the lower cervical and upper thoracic regions (right side) and erosion of the first and second thoracic ribs and transverse process of the seventh cervical vertebra.

Involvement of the brachial plexus may occur in infectious neuritis such as may be associated with infectious diseases like diphtheria, scarlet fever, typhoid fever or other systemic infections, such as tuberculosis and syphilis. However, there is no convincing evidence that such foci of infection as may be found in the teeth, the prostate, the gall bladder or the appendix will give rise to pains in the extremities by producing agents (toxins) which act directly on the nerve tissue.

Clinical investigation discloses that the greater majority of cases exhibiting this symptom complex fall into one of the three following groups: (1) Lesions of the musculotendinous cuff; (2) protrusion of intervertebral disks in the cervical region; and (3) bicipital tenosynovitis.

Localized suppurative processes in the supraclavicular and the axillary regions may implicate the nerve by direct extension.

SERUM NEURITIS

Varying degrees of sensory and motor disturbances occasionally arise following injection of sera. Signs and symptoms of nerve involvement manifest themselves about two days after the onset of serum

tion of the costoclavicular space and compression of the subclavian vessels. Resection of portion of the first thoracic rib at the site of maximum compression relieved the patient of all symptoms.

Moreover hyperextension of the neck and backward and downward displacement of the shoulders no longer produced obliteration of the pulse. The above observers and Walshe, Jackson and Wyburn Mason called attention to the numerous anatomic and developmental factors which may predispose to compression by the costoclavicular mechanism. Notably among these are a high first thoracic rib, high fixed sternum, abnormal descent or sagging shoulders and abnormal thoracic operculum.

It is obvious that tests (such as hyperextension of the neck, turning the head to one side or the other together with deep respiration, hyperabduction of the arms and depressing the shoulder girdle) hitherto used for scalenus anticus and cervical rib syndrome are no longer useful diagnostic aids. As has been shown these same tests produce phenomena of obliteration of the subclavian artery in normal individuals. Also, it has been pointed out that several mechanisms can produce the same compression phenomena.

TREATMENT

Adequate evaluation of the symptom complex is essential before therapy is instituted. Simple scalenotomy may result in a high percentage of failures if done indiscriminately.

The presence of a cervical rib associated with well-developed manifestations of nervous compression and vascular destruction is conclusive evidence that the anomalous rib is the prime operative agent. Operative intervention is indicated in these instances. Division of the scalenus anticus alone may relieve the symptoms, however resection of varying portions of the cervical rib may also be necessary. In other instances especially in those in which no abnormality of the costal elements or thoracic outlet is

demonstrable by roentgenograms, conservative measures should be instituted. These comprise elimination of activities which tend to place the arm in hyperabduction, keeping the arms below the horizontal position during sleep, making the patient posture-conscious by insisting that the shoulders be maintained in an elevated position at all times, exercises to improve the tone of the muscles of the shoulder girdle, and general measures to improve the economy as a whole.

In cases with severe symptoms, failure to respond to a conservative regimen justifies surgical exploration of the supraclavicular area in search of the cause responsible for the compression phenomena. Again it must be emphasized that surgical intervention is warranted only after all possible etiologic factors have been duly considered.

TUMORS

Primary and secondary metastatic neoplasms may involve the brachial plexus and may produce radicular pain and signs and symptoms of vascular compression in the upper extremity. Primary bronchogenic carcinoma of or metastatic lesions to the apices of the lungs may implicate the neurovascular structure in the supraclavicular and the cervical regions by direct extension. Not infrequently carcinoma of the breast even after a radical mastectomy will invade the brachial plexus. Lymphosarcoma and Hodgkin's disease involving the lymph nodes in the supraclavicular and the cervical regions may produce the syndrome.

Pancoast in 1924 described an apical lung tumor which he designated superior pulmonary sulcus tumor. The derivation of this tumor is not clear. Pancoast expressed the belief that it was of epithelial origin. Its characteristic features are pain referred to the shoulder region, the axilla and down the ulnar aspect of the arm, atrophy and weakness of the muscles of the hand chiefly the interossei and the hypothenar muscles, Horner's syndrome and roentgenographic evidence of a pulmonary mass in the apex.

of the lung, with evidence of erosion of the upper ribs, the transverse processes and in some instances the vertebral bodies (Fig 287)

Some investigators maintain that the sympathetic manifestations (Horner's syndrome) are the result of pressure on the sympathetic fibers in the common trunk from the eighth cervical and the first thoracic. Others contend that they result from involvement of the stellate ganglion which lies just anterior to the head and the neck of the first rib, or slightly below this level. Sensory and motor manifestations pointing to involvement of the eighth cervical and the first and the second thoracic nerve roots and Horner's syndrome are the principal features of this symptom complex.

Neoplasms arising from the sheath cells of the nerves comprising the brachial plexus anywhere along their course, may be responsible for pain projected to the shoulder or the arm.

Neurinoma commonly arise from the cervical and the thoracic roots of the spinal nerves. They produce segmental, sensory and motor phenomena and exhibit such characteristic features of root involvement as exacerbation of pain by increased intra thoracic or intra abdominal pressure and by maneuvers designed to stretch the nerve roots. As a rule neurinomas appear as single lesions. However they may be multiple particularly if associated with generalized neurofibromatosis. Single neurofibromas occasionally arise from the nerves of the brachial plexus and may be demonstrable by palpation of the cervical, the supraclavicular or the axillary regions. The brachial plexus may be implicated in generalized neurofibromatosis.

INFLAMMATORY LESIONS

Increasing knowledge of the numerous factors that may account for neck, shoulder and arm pain has reduced considerably the number of heterogeneous entities hitherto grouped under the all inclusive term "brachial plexus neuritis".



FIG 287 Roentgenogram of the thoracic outlet of a case of lancost tumor. Observe the soft tissue mass in the lower cervical and upper thoracic regions (right side) and erosion of the first and second thoracic ribs and transverse process of the seventh cervical vertebra.

Involvement of the brachial plexus may occur in infectious neuritis, such as may be associated with infectious diseases like diphtheria, scarlet fever, typhoid fever or other systemic infections, such as tuberculosis and syphilis. However, there is no convincing evidence that such foci of infection as may be found in the teeth, the prostate, the gall bladder or the appendix will give rise to pains in the extremities by producing agents (toxins) which act directly on the nerve tissue.

Clinical investigation discloses that the greater majority of cases exhibiting this symptom complex fall into one of the three following groups: (1) Lesions of the musculotendinous cuff, (2) protrusion of intervertebral disks in the cervical region and (3) bicipital tenosynovitis.

Localized suppurative processes in the supraclavicular and the axillary regions may implicate the nerve by direct extension.

SERUM NEURITIS

Varying degrees of sensory and motor disturbances occasionally arise following injection of sera. Signs and symptoms of nerve involvement manifest themselves about two days after the onset of serum

sickness. Although other peripheral nerves may be affected, there appears to be a predilection for the upper components of the brachial plexus. The injection site is in no way related to the specific nerves implicated. Cases of serum neuritis of the brachial plexus have been reported following injection into the abdominal wall. Implication of the axillary nerve is a common sequel.

Evidence of the neuritis is first noted by intense pain in the shoulder region and along the outer aspect of the arm. Sensory and motor phenomena appear following a definite segmental pattern pointing to involvement of specific cervical nerve roots, usually the fifth and the sixth. Muscular weakness, atrophy, decreased muscle reflexes and paralysis are the outstanding motor signs. The muscles most commonly affected are the deltoid, the biceps, the brachialis and the brachioradialis, all innervated by the upper elements of the brachial plexus. Recovery is the rule, the period for complete recovery depending upon the regeneration time required by the affected nerve. The pathologic disorder responsible for the neuritis is believed to be a localized inflammatory process involving the perineurium which compresses the nerve fibers.

TRAUMATIC LESIONS

Trauma to the brachial plexus and its peripheral nerves accounts for pain and disability of the upper extremity in a large number of cases. According to Grinker and Bucy, 10 per cent of all traumatic cases in civil life are due to birth injuries and 12 per cent to the automobile. Injuries which depress the shoulder or increase the angle between the shoulder and the neck are responsible for many brachial lesions (partial or complete). Direct pressure into the axilla (crutch paralysis) is another form of injury. Too often the position of the upper extremities of patients on the operating table is responsible for nerve lesions due to prolonged traction and torsion of the plexus. Penetrating and lacerating wounds in the

supraclavicular, the infraclavicular and the axillary regions are apt to produce bizarre syndromes. This has been amply demonstrated by war injuries to these regions.

Other types of minor traumata may also be operative factors in producing brachial plexus lesions. Chief among these are repeated traumata to nerves, sustained during the performance of certain occupations. Fracture dislocations and dislocations of the shoulder joint are complicated not infrequently by injuries to the brachial plexus or its branches.

OBSTETRIC PARALYSIS

INTRODUCTION

Three types of obstetric paralysis are recognized clinically: (1) the upper-arm or Erb-Duchenne type, (2) the lower-arm or Klumpke type, and (3) the whole-arm type.

Immediately after birth the extremity usually exhibits the clinical features of flaccid paralysis, implicating the entire limb. The arm assumes a characteristic position: it is kept extended at the side of the body, the upper arm rotates internally, the forearm is pronated, forcing the palm to be directed backward and outward, and the wrist may be flexed slightly. In mild forms there may be some voluntary motion in the fingers, the hand, the wrist and the elbow. No motor power is noted in any part of the limb in the more severe cases. Fortunately, evidence of return of motor power is manifested in most cases within a relatively short period of time. Voluntary motion starts first in the fingers, then in the hand, the wrist and the elbow. Spontaneous recovery continues until the level of permanent nerve injury is reached, where no recovery is possible. The group of muscles paralyzed corresponds to the roots implicated. Many cases of palsy are associated with injuries to the osseous components of the joint, which may not be evident at birth but may be demonstrable radiographically as early as 3 weeks later. Fractures near or through the epiphyseal plate may

occur, the proximal humeral epiphysis may be traumatized or displaced for varying distances on the metaphysis. Posterior subluxation of the epiphysis is commonly encountered in the more severe types. Such injuries favor maldevelopment of the epiphysis and of the glenoid cavity and they cause marked impairment of function, even in the presence of good motor power.

ETIOLOGY AND PATHOGENESIS

There is general agreement that trauma inflicted on the child during its exit from the birth canal plays a major role in the production of these palsies. The belief is substantiated by the following clinical facts:

- 1 Lesions are associated most frequently with prolonged and difficult labor.

- 2 A large proportion of the children affected are delivered by forceps.

- 3 First borns are affected more frequently.

- 4 Breech presentations show a higher percentage of paralytic conditions than vertex presentations.

On the other hand there are many divergent concepts as to the pathogenesis of obstetric paralysis. Many observers postulate that there is direct injury to the component parts of the brachial plexus. The nerve trunks are overstretched by forceful separation of the head and the shoulders. This results in injury to the nerves in continuity (nerve fibers are frayed within the sheath) or by rupture of the roots. Rupture may occur anywhere along the trunks or the roots may be avulsed from the spinal cord. The number of roots affected and the severity of the lesions vary considerably; generally the upper roots are implicated first and sustain more severe injury than the lower roots. In addition to the intrinsic nerve injuries usually there are varying degrees of soft tissue damage such as tearing of the deep cervical fascia and the nerve sheaths, rupture of blood vessels and hemorrhage and exudation from traumatized structures. In the subsequent healing process the nerves may be entangled in a

mass of contracting scar tissue. This offers a formidable barrier to nerve regeneration. Supporters of this premise believe that shoulder deformities, particularly subluxation of the head of the humerus, are secondary manifestations due to contracture of unopposed muscles.

Taylor was unable to demonstrate a posterior dislocation in children under 3 weeks old although this deformity progressive in nature was discernible in children after 3 weeks. In a number of additional cases operated upon, Taylor and others noted complete rupture of the upper roots of the brachial plexus and in some actual avulsion of the roots from spinal cord.

T. T. Thomas was in total disagreement with the above mentioned theory. He was of the opinion that the prime etiologic factor is injury to the shoulder joint structures, particularly the capsule and posterior subluxation of the humeral head at the time of birth. He believed that extravasation of blood and tissue fluids into the axilla set up an inflammatory process which subsequently involved the brachial plexus and produced paralysis, followed by fibrous tissue formation which in due time is absorbed so that paralysis gradually disappears. He believed also that at the time of injury the acromion process is bent downward in front of the humeral head, preventing spontaneous repositioning of the head in the glenoid cavity and maintaining it in a fixed subluxated position.

He maintained moreover that very rarely was actual nerve rupture a concomitant lesion of the paralysis. In support of this concept he offered the following pertinent clinical observations:

- 1 There is almost uniform and gradual disappearance of the palsy.

- 2 Dysfunction of the limb in adults is not the result of paralysis but of a posterior dislocation of the shoulder.

- 3 Immediately after birth the paralysis of the limb is extensive and not limited to a specific group of muscles.

- 4 Correction of the deformity of the

shoulder is invariably followed by improvement of function

In the light of the work in support of the two theories discussed above, it is more reasonable to conclude that some cases of

TREATMENT

Adequate treatment should be instituted immediately after birth. The extremity is placed in a position of abduction and external rotation to prevent adduction and

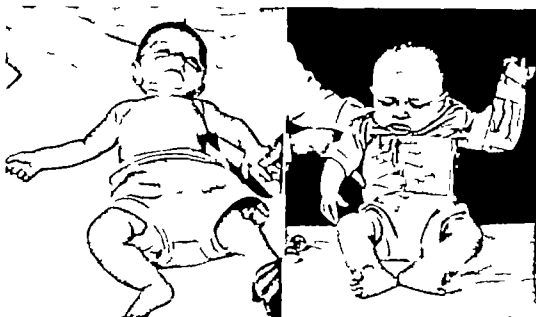


FIG. 288 (*Left*) Child with the upper arm form of obstetrical palsy (Erb-Duchenne). The forearm is extended and pronated; the upper arm is rotated inward and cannot be abducted. (*Right*) Abduction brace for obstetrical paralysis.

obstetric palsy are the result of direct injury to the brachial plexus with subsequent true paralysis of specific groups of muscles; others are pseudoparalysis caused by shoulder joint trauma with deformity simulating true paralysis while in still others both concepts may be applicable.

Older children invariably reveal clinically and radiographically changes in the bony architecture of the joint. Varying degrees of posterior subluxation of the head of the humerus may exist; in such cases the acromion is usually elongated and bent forward and downward in front of the humeral head; the coracoid process also may be elongated and crooked in front of the head; torsion of the humeral shaft is demonstrable after the age of 3; the humeral epiphysis is underdeveloped and malshaped; the scapula rides high and is rotated inward; and finally the glenoid cavity is flattened.

flexion deformities of the shoulder and internal rotation of the humerus. This is best achieved by a light splint which fixes the arm in the coronal plane 90° abduction and external rotation; the forearm is flexed 90° and fully supinated; the wrist is dorsiflexed (Fig. 288). Remarkable recovery of motor power is discernible in the first 3 months in the great majority of cases. Gentle passive stretching of the shoulder in external rotation and abduction with the forearm in supination is a valuable adjunct to the treatment. If the child tends to use the unaffected arm more than the affected arm while the brace is off, the good extremity should be tied or pinned to the side to promote more use of the impaired limb. These measures must be strictly executed.

Conservative measures are employed in the upper-arm type to prevent contractures and to re-educate weakened muscles. Ex-

ploration of the brachial plexus seldom has much to offer but may be attempted especially if the paralysis is limited to a specific group of muscles. Sever recommended waiting until the fourth or the fifth year before contemplating surgical intervention, at which time severance of contracted soft tissue structures may be done also. Although the results are usually disappointing some observers (Taylor for example) recommend exploration of the brachial plexus at the end of three months in cases showing no spontaneous recovery, particularly in the whole arm type.

In older children with posterior dislocation of the shoulder the muscle-cutting operation of Sever and elevation or removal of the acromion process improves both the function and the cosmetic appearance of the extremity.

TYPES OF OBSTETRIC PARALYSIS

Upper Arm or Erb-Duchenne Type. This lesion is encountered most frequently of the three types of obstetric palsies. The fifth and the sixth cervical roots are involved thereby causing paralysis of the deltoid, the biceps, the brachialis and the supinator longus muscles. Occasionally, the supraspinatus, the infraspinatus, the subscapularis and the supinator brevis muscles and the extensors of the wrist and the fingers may be implicated.

The condition is produced by spreading the interval between the head and the shoulder (such as occurs when traction is made on the head at birth) or by forcing the shoulder downward and backward. The limb assumes a characteristic attitude: both the arm and the forearm are adducted and internally rotated; the forearm is extended and pronated. Lesions of long duration exhibit marked atrophy of the deltoid, the biceps, the brachialis and the supinator muscles. Motion is greatly impaired. There is loss of abduction and external rotation of the arm and loss of flexion and supination of the forearm.

A pertinent clinical feature is the lack of sensory disturbances except for a small patch of loss of sensation over the posterolateral aspect of the shoulder. This can be explained readily by the extensive overlap of sensory fibers of intact nerve roots. Such is not the case in complete lesions implicating the entire plexus, in which there is profound sensory loss—in fact complete anesthesia of the upper extremity. Generally, with correct management in obstetric upper arm palsies the prognosis for partial or complete recovery is favorable.

Lower Arm or Klumpke Type. This lesion is indeed rare. It implicates the eighth cervical and first thoracic nerve roots producing paralysis of the muscles which are supplied by the ulnar and inner head of the median nerves. The intrinsic muscles of the hand and the flexors of the forearm are involved. Cervical sympathetic nerve fibers traversing the lower two nerve roots may be injured producing pupillary inequality (the pupil on the affected side is reduced in size). This is a rare concomitant finding. As a rule the prognosis for recovery is poor. If such an abnormal sympathetic manifestation persists.

The lesion is produced by forcing the extremity upward, hence making tension on the lower roots of the plexus (such as is caused from traction made in breech presentations with the arm extended). Atrophy of the paralyzed muscles and incomplete closing of the hand occurs, and sensory loss is demonstrable in the dermatomes of the eighth cervical and first thoracic nerve roots.

Whole Arm Type. This lesion is characterized by implication of the entire plexus: there is flaccid paralysis of the entire extremity and the wrist paralysis of the small muscles of the hand and complete sensory loss. Many gradations in severity occur between the upper arm and the whole arm types depending upon the extent of the injury and the number of roots affected. Both extremities may be involved.

OPERATIVE PROCEDURES FOR OBSTETRIC PARALYSIS

Severs operation is the most popular means of relieving the adduction and internal contractures in cases of obstetric paralysis. Optimum restoration of function depends upon proper selection of cases and adequate physical therapy in muscle development prior to operation.

The procedure should not be performed before the age of 4 or 5 years and yields the best results when posterior subluxation or posterior torsion of the head of the humerus are not associated lesions. The best func-

tional results are attained in cases which exhibit pronounced dysfunction of the shoulder, cases with slight limitation of motion are not benefited sufficiently to warrant surgical intervention.

Severs's Technic. An anterior skin incision is made beginning at the tip of the acromion to below the insertion of the pectoralis major muscle. The deltopectoralis cleft is developed, taking care not to injure the cephalic vein. By retracting the deltoid laterally, the tendinous insertion of the pectoralis major comes into view and is severed close to its insertion.

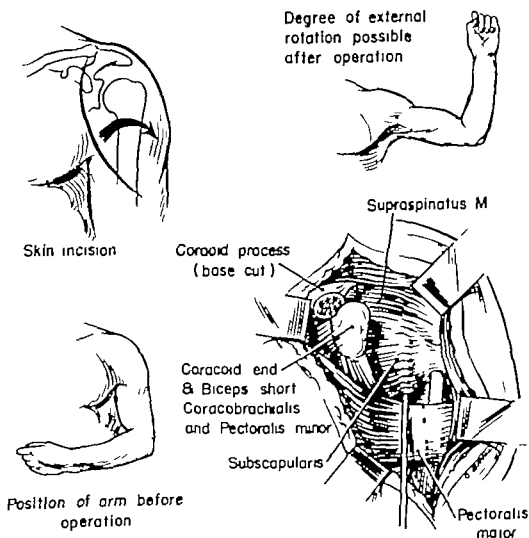


FIG 289 Sever's operation for obstetrical paralysis. (Top left) Skin incision. (Top right) After operation the arm can be adducted readily and rotated externally. (Bottom left) Before operation the arm is held to the side adducted and rotated internally. (Bottom right) Division of the pectoralis major and the subscapularis tendon. The tip of the coracoid is removed together with the short head of the biceps the coracobrachialis and the pectoralis minor.

Medial retraction of the pectoralis major with the extremity abducted and rotated externally uncovers the coracobrachialis muscle, which is traced to its origin from the coracoid process. Generally the tip of the coracoid is found to be elongated and crooked downward in front of the head of the humerus its tip (together with the insertion of the coracobrachialis the short head of the biceps and the pectoralis minor muscles) is osteotomized at its base and removed. Approximately one quarter to three eighths of the tip is removed. With retraction of the coracobrachialis and the short head of the biceps downward and removal of the tip of the coracoid process considerable gain is achieved in abduction and external rotation.

Next the tendon of the subscapularis muscle is identified as it inserts into the lesser tuberosity. A curved director is passed beneath the tendon from below upward and the tendon is divided completely across, without cutting the fibrous capsule. Division of this tendon allows optimum elevation abduction and external rotation of the humerus.

Complete abduction may be impeded by elongation and hooking of the acromion. Resection of a portion of the end of the acromion will remove the bony block. To obtain complete abduction in old and severe contractures it may be necessary (in rare cases) to divide the insertion of the latissimus dorsi and the teres major muscles through an approach along the posterior axillary fold (Steindler).

POSTOPERATIVE MANAGEMENT

Following operation the arm is immobilized in an abduction plaster-of-Paris shoulder spica it is maintained in abduction and external rotation for from 10 to 14 days. The cast is then removed and an abduction brace is applied to hold the arm in the above position. Essential features of the postoperative regimen are physical therapy, in the form of massage stretching active

and passive motion and particularly muscle re-education.

This procedure gives uniformly satisfactory results in the majority of cases of obstetric paralysis involving the upper arm. The arm is still far from normal but its function is greatly enhanced.

OSTEOTOMY OF THE HUMERUS AND MUSCLE RELEASE

In many children exhibiting adduction and internal rotation contracture of the shoulder no evidence of muscle paralysis is demonstrable. T. T. Thomas believed that such lesions were primarily those of injury to the shoulder joint at the time of birth, followed by an inflammatory process in the axilla which produced a pseudoparalysis. With healing and absorption of the inflammatory tissue the paralysis disappears. He also believed that posterior subluxation of the head of the humerus and bending of the acromion process occurred at the time of the initial injury. When maintained in a position of posterior subluxation by contracture of the adductors and the internal rotators of the humerus and the bent acromion the humeral head undergoes varying degrees of posterior torsion which precludes replacement and maintenance of the head in the glenoid cavity by simple division of the subscapularis and the pectoralis major muscles.

In these cases repositioning and maintenance of the humeral head in the glenoid cavity is achieved best by the addition to the Sever operation of an osteotomy through the surgical neck of the humerus.

Operative Technic (Scaglietti) The first step consists in the correction of the adduction and internal rotation deformity by Sever's operation as described above. The head of the humerus is thus restored to its normal anatomic relationship in the glenoid fossa. The extremity is then placed and maintained in abduction and external rotation for 8 weeks. Next through the lower end of the anterior incision a transverse osteotomy is done through the surgical neck

of the humerus. While the upper head portion of the humerus is held in its normal position, the distal portion is rotated internally.

Postoperative Management. Immobilization of the extremity in a plaster-of-Paris shoulder spica is maintained until bony union occurs. Removal of the cast is followed by intensive physical therapy particularly muscle re-education.

Osteotomy of the Humerus. Simple osteotomy to correct internal rotation deformities through the surgical neck of the humerus may be performed in cases exhibiting no posterior torsion or subluxation of the head of the humerus (Vulpius, Lange, Rogers). Although the cosmetic results are desirable, the improvement in function does not justify the procedure. From a functional viewpoint, Sever's operation in these cases is far superior to osteotomy of the humerus.

Correction of Internal Rotation Deformity and Posterior Subluxation of Humerus by Reattachment of Capsule

and External Rotators (Kleinberg) Kleinberg designed the operation for correction of internal deformities of the humerus associated with posterior torsion and posterior subluxation of the humeral head. Essentially, it consists of a subperiosteal detachment of all the rotators and the capsule from the upper 1 to 1½ inches of the humerus. Two flaps are created: a lateral flap comprising the external rotators and fibrous capsule and a medial flap, consisting of the subscapularis muscle and fibrous capsule. Freed of all muscular and capsular attachment, the humerus is readily rotated externally. The lateral flap is drawn more medially and is sutured in this position; it is overlapped by the medial flap.

Correction is achieved and maintained by this procedure. However, extensive stripping of all soft tissues from the upper end of the humerus is likely to cause vascular damage resulting in degenerative changes and disturbance in growth of the upper end of the humerus. Kendrick has recorded such undesirable sequelae in the literature.

BIBLIOGRAPHY

- Adson, A. W. Surgical treatment of cervical ribs. *Texas State J. Med.* 28: 39-42, 1933.
- Adson, A. W. and Caffey, J. B. Cervical rib: a method of anterior approach for relief of symptoms by division of the scalenus anticus. *Ann. Surg.* 85: 839-85, 1927.
- Braish, J. C., and Jamieson, E. B. *Cunningham's Text Book of Anatomy*, ed. 8. New York, Oxford Univ. Press, 1943, p. 1558.
- Broders, A. C. The grading of carcinoma. *Minnesota Med.* 8: 726-730, 1925.
- Bucy, P. C., and Chenault, H. Compression of seventh cervical nerve root by herniation of an intervertebral disk. *J.A.M.A.* 126: 26-27, 1944.
- Codman, E. A. *The Shoulder*. Boston: Thomas Todd, 1934, p. 513.
- Craig, W. McK., and Knepper, P. A. Cervical rib and the scalenus anticus syndrome. *Ann. Surg.* 105: 556-563, 1937.
- Eaton, L. M. Pain caused by disease involving the sensory nerve roots (root pain): its characteristics and mechanics of its production. *J.A.M.A.* 117: 1435-1439, 1941.
- Eden, K. C. The vascular complications of cervical ribs and first thoracic rib abnormalities. *Brit. J. Surg.* 27: 111-139, 1939.
- Elliott, F. A., and Kremer, M. Brachial pain from herniation of cervical intervertebral disk. *Lancet* 1: 4-8, 1945.
- Falconer, M. A., and Weddell, G. Costoclavicular compression of the subclavian artery and vein. *Lancet* 2: 539-543, 1943.
- Grant, J. C. B. *A Method of Anatomy*. Baltimore: Williams & Wilkins, 1940, p. 194.
- Haymaker, W., and Woodhall, B. *Peripheral Nerve Injuries*. Philadelphia: Saunders, 1945, p. 227.
- Hill, R. M. Vascular anomalies of the upper limbs associated with cervical ribs: report of a case and review of the literature. *Brit. J. Surg.* 27: 100-110, 1939.
- Inman, V. T., and Saunders, J. B. Referred pain from skeletal structures. *J. Nerv. and Ment. Dis.* 99: 660-661, 1944.
- Jaffe, H. L. "Osteoid-osteoma": benign osteoblastic tumor composed of osteoid and atypical bone. *Arch. Surg.* 31: 69-728, 1935.
- Jaffe, H. L. Osteoid osteoma. *Am. J. Path.* 12: 796, 1936.

- Jaffe H L and Lichtenstein L. Osteoid osteoma: further experience with this benign tumor of bone. *J Bone & Joint Surg* 22 645-682 1940
- Jones F W. Variations of the first rib associated with changes in the constitution of the brachial plexus. *J Anat* 45 249-255 1911
- Jones F W. Discussion on cervical ribs: the anatomy of cervical ribs. *Proc Roy Soc Med* 6 95-113 1911
- Lewis T. *Pain*. New York: Macmillan 1942 p 192
- Meyerding H W. The preoperative differential diagnosis of bone tumors. *J.A.M.A.* 88 365-371 1927
- Meyerding H W. The results of treatments of osteogenic sarcoma. *J Bone & Joint Surg* 20 933-948 1938
- Meyerding H W. Classification of bone tumors. *Proc. Staff Meet., Mayo Clin* 18 1-18 1943
- Meyerding H W. Chronic sclerosing osteitis. *S Clin. North America* 24 62-9 1944
- Meyerding H W and Valls J E. Primary malignant tumors of bone. *J.A.M.A.* 117 237-243 1941
- Michelsen J J and Mixer W J. Pain and disability of shoulder and arm due to herniation of the nucleus pulposus of cervical intervertebral disks. *New England J Med.* 231 279-287 1944
- Mosely H W. *Shoulder Lesions*. Thomas Springfield, Ill 1945
- Nachlas I W. Brachialgia: manifestations of various lesions. *J Bone & Joint Surg* 26 177-184 1944
- Naffziger H C and Boldrey E B. Surgery of spinal cord. In Bancroft F W and Pilcher C., *Surgical Treatment of the Nervous System*. Philadelphia: Lippincott 1946 pp. 327-406
- Naffziger H C and Grant W T. Neuritis of the brachial syndrome: plexus mechanical in origin: the scalenus syndrome. *Surg Gynec. & Obst* 67 722-730 1938
- Olsen A M. The role of thoracic disease in the production of arm pain. *S Clin North America* 26 801-803 1946
- Pancoast, H K. Superior pulmonary sulcus tumor: tumor characterized by pain: Horner's syndrome: destruction of bone and atrophy of hand muscles. *J.A.M.A.* 99 1971-1976 1932
- Pfemister D B. Rapid repair of defect of femur by massive bone grafts after resection for tumors. *Surg Gynec & Obst* 80 120-127 1945
- Semmes R E and Murphey F. Syndrome of unilateral rupture of sixth cervical intervertebral disk with compression of seventh cervical nerve root. *J.A.M.A.* 121 1209-1214 1943
- Smith H J. Arm pain due to heart disease. *S Clin North America* 26 504-505 1946
- Stookey B. Compression of the spinal cord due to central extradural cervical chondroma: diagnosis and surgical treatment. *Arch. Neurol. & Psychiat* 20 275-291 1928
- Stookey B. Compression of spinal cord and nerve roots by herniation of nucleus pulposus in cervical regions. *Arch. Surg* 40 417-432 1940
- Stopford, J S B., and Telford, E. D. Compression of the lower trunk of the brachial plexus by a first dorsal rib: with a note on the surgical treatment. *Brit J Surg* 7 163-177 1919
- Swank R. L., and Simeone F A. The scalenus anticus syndrome: types, their characterization, diagnosis and treatment. *Arch. Neurol. & Psychiat* 51 432-445 1944
- Todd, T W. The descent of the shoulder after birth. *Anat. Anz.* 41 385-397 1912
- Todd, T W. Posture and the cervical rib: a method of anterior approach for relief of symptoms by division of the scalenus anticus. *Ann. Surg* 85 839-857 1927
- Wallman, H. W. *Neuritis in Tice Frederick Practice of Medicine*. Vol. 9 chap. 3 pp 289-414 Hagerstown Md. Prior 1923
- Walshe F M R., Jackson H and Wyburn M R. On some pressure effects associated with cervical and rudimentary and "normal" first ribs, and the factors entering into their causation. *Brain* 67 141-177 1944
- Wartenberg, R. Brachialgia statica paresthetica (nocturnal arm dysaesthesia). *J Nerv. & Ment. Dis* 99 877-887 1944
- Wright I S. The neurovascular syndrome produced by hyperabduction of the arms. *Am Heart J* 29 1-19 1945

10

Bone Tumors of the Shoulder Joint

DIAGNOSIS OF BONE TUMORS

GIANT CELL TUMOR

EPIPHYSEAL CHONDROMATOUS GIANT-CELL TUMOR

SOLITARY BONE CYST

SARCOMAS OF THE BONE

OSTEOGENIC SARCOMA

SCLEROSING OSTEOGENIC SARCOMA

TELANGIECTATIC SARCOMA OF THE BONE

MEDULLARY AND SUBPERIOSTEAL OSTEOGENIC SARCOMA

FIBROSARCOMA OF THE BONE

EWING'S SARCOMA

OSTEOID OSTEOMA

CHONDROSARCOMA

CHONDROMA

OSTEOCHONDROMA

MULTIPLE MYELOMA

SOLITARY PLASMA CELL MYELOMA

METASTATIC CARCINOMA IN THE BONES OF THE SHOULDER GIRDLE

MYOSITIS OSSIFICANS CIRCUMSCRIPTA

GENERAL CONSIDERATIONS

INTRODUCTION

Neoplasms benign and malignant are encountered frequently in the shoulder region. Codman's Survey of the Registry of Bone Sarcoma (1922) disclosed that 13.5 per cent of all bone tumors were found in the bones of the shoulders, osteogenic sarcoma of the head of the humerus being most commonly found.

The noted frequency of tumors in this region makes it imperative to discuss them as a group, to sharpen our focus in the diagnosis and the treatment of shoulder disorders. This chapter does not aim to present in detail the many controversial theories of histology and origin of tumors but is concerned primarily with the recognition and the management of such lesions. Generally tumors of the shoulder do not differ from those in other parts of the body. However it should be recognized that accurate diagnosis and adequate treatment based on sound clinical and surgical judgment are more important in managing lesions in this than other parts of the body. An error here

may mean the loss of a highly specialized extremity and seriously impair the patient's economic status.

As in lesions affecting other parts of the skeleton lesions in the bones of the shoulder girdle must be investigated thoroughly to establish their identity. The skill and the rapidity with which this is done may have a direct bearing upon the ultimate outcome of the case. Even today when both laymen and physicians are reminded constantly of the value of early diagnosis and treatment of cancer many instances of cancer come to a sad end because the patient or the physician fails to bring about an early diagnosis and adequate therapy.

CLASSIFICATION OF BONE TUMORS

Classification of tumors of bone remains a confusing problem. This stems from a lack of agreement among investigators on the basis of a classification. Workers approach the perplexing subject from divergent viewpoints: some attempt to classify neoplasms and other bone lesions on their anatomic and histologic characteristics; others believe that a classification should denote

CLASSIFICATION OF BONE TUMORS (COLEY)

Malignant

Fibrosarcoma of bone
Osteogenic sarcoma

Primary Chondrosarcoma
Secondary chondromyxosarcoma
Malignant giant-cell tumor

Endothelioma (Ewing's sarcoma)
Angiosarcoma

Myeloma { plasma-cell myeloma
 myelocytoma
 erythroblastoma
 lymphocytoma
Reticulum-cell sarcoma
Liposarcoma

Benign

Nonosteogenic fibroma of bone
Osteoma
Osteoid Osteoma
Exostosis
Chondroma

Benign giant-cell tumor
Benign chondroblastoma (Coulman's epiphyseal chondromatous (giant-cell tumor)
Unicameral bone cyst
Cavernous angioma
Plexiform angioma

pathologic or radiographic features of the lesions.

Happily however there is a trend to crystallize a classification of primary bone neoplasms in accordance with the predominant cell or tissue found in the tumor and arising from the undifferentiated germ cells of bone. This trend is being carried still further to include tumors from the accessory tissues of bone (bone marrow, reticulo-endothelial system and marrow connective tissue).

Coley modified the revised classification of the Registry of Bone Sarcoma of the American College of Surgeons to include benign and malignant neoplasms.

This revised classification has placed malignant neoplasms derived from cartilage in a separate group. The osteogenic group is subdivided further in accordance with the location and the radiographic features of the tumors. The subdivisions are

- 1 Medullary and subperiosteal
- 2 Telangiectatic
- 3 Sclerosing
- 4 Periosteal
- 5 Fibrosarcoma
 - A. Medullary
 - B. Periosteal
- 6 Capsular and parosteal

DIAGNOSIS OF BONE TUMORS

HISTORY

A carefully taken history may give information of great value in arriving at a correct diagnosis. In many instances it will establish the diagnosis, in others it will contribute important data which together with other diagnostic aids, will permit the diagnosis to be made. Rapidly growing tumors regardless of their location or the patient's age must be considered very malignant. Slowly growing tumors are less malignant. Increase in size of a tumor previously existing for a long time without evidence of activity is usually indicative of secondary malignant changes in a benign lesion.

AGE OF PATIENT

Age is always an important item in evaluating bone tumors particularly osteogenic sarcoma. Badgley and Batts noted that the greatest number of osteogenic sarcomas occur in the second decennium (40 of 80 cases reported). According to Christensen two-thirds of 441 cases of bone sarcoma occurred between 10 and 30 and one third over 30 years also, that very young and

very old people are rarely victims of bone sarcoma

Codman emphasized that osteogenic sarcoma in patients around 50 is usually indicative of Paget's disease with secondary sarcomatous changes. In a review of 17 sarcomas of the upper end of the humerus listed in the Bone Sarcoma Registry he found 3 cases around 50 years of age. All three had Paget's disease of the bone. Bird records that 14 per cent of all cases of Paget's disease develop bone sarcoma. Bone cysts occur before closure of the epiphyseal disk, while giant-cell tumors are found after the epiphysis has united. Plasma-cell myeloma is rarely encountered under the age of 40, metastatic lesions are usually found past middle life.

PAIN

In the early stages of a malignant bone neoplasm pain is an exceedingly variable symptom. It may be mild and present only on activity or intermittent so that its seriousness is not appreciated by the patient or the physician. As a rule pain comes on insidiously. Occasionally its onset is sudden and may be accompanied by a systemic reaction similar to an infectious process such as osteomyelitis. This is particularly true of Ewing's tumor and highly malignant rapidly growing osteogenic sarcomas.

Later the pain becomes more constant and often is referred to as boring in character. It is accentuated by activity and is more pronounced at night. Usually it is localized over the affected portion of the bone; occasionally it is referred to areas distal to the site of the lesion. Pain in malignant lesions of the upper end of the humerus is referred not infrequently to the region of the deltoid insertion or to the inferior angle of the scapula. Expansile lesions such as giant-cell tumors of the upper end of the humerus are accompanied often by a steady constant dull deep-seated pain.

Benign tumors are rarely painful unless they interfere with the excursion of muscles

overlying them or act as a mechanical barrier to normal joint motion. In many instances, the first indication of the existence of a benign cystic lesion may be fracture of the bone. Giant-cell tumors are the exception, pain of varying degrees is usually present. Primary malignant tumors usually are accompanied by pain, while metastatic lesions may fail to produce pain.

INJURY

In a large group of cases trauma antedates the symptoms and the local findings. Its significance is of doubtful value, except that it may serve to make the patient aware of an existing lesion. It is generally accepted that a single injury, regardless of the degree of severity, is not the prime causative agent responsible for the development of malignant neoplasms of bone. A detailed account of the trauma, e.g., its time and manner of occurrence, severity and demonstrable local evidence of injury at the time of examination should be noted and recorded. Such data may be of importance should the case assume medicolegal proportions.

LOSS OF FUNCTION

Tumors in the region of the shoulder rarely produce marked disability except in the late stages of the disease. Large and rapidly growing lesions of the scapula and the upper end of the humerus may be accompanied by spasm and contracture of the adductor muscles, e.g. the pectoralis major and minor, the latissimus dorsi and the teres major muscles. This results in marked restriction of glenohumeral and scapulohumeral motion and total disruption of the capulohumeral rhythm.

PAST HISTORY

Past history will elicit the presence or the absence of repeated trauma and strains to the involved area, also the previous existence of infectious processes which might primarily or secondarily affect the osseous system. Notably among the infectious processes are tuberculosis, osteomyelitis, syph-

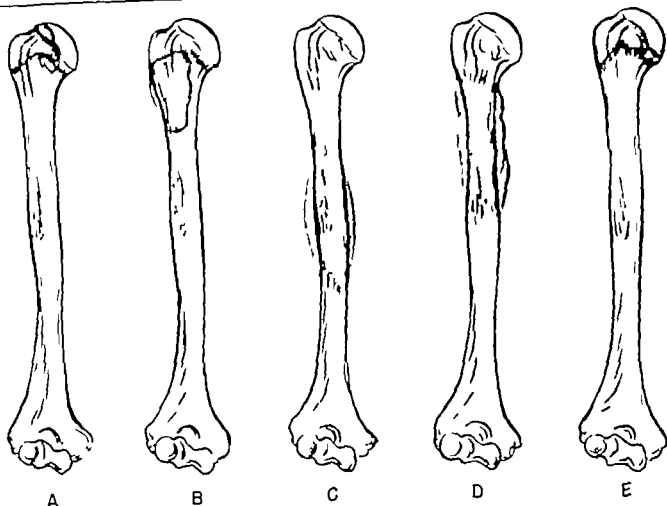


FIG. 290 Common sites of primary tumors in the upper end of the humerus. (A) Giant-cell tumor (B) Bone cyst (C) Ewing's tumor (D) Osteogenic sarcoma. (E) Chondrosarcoma.

this typhoid fever and brucellosis. Metabolic and deficiency disorders such as gout, rickets and scurvy are capable of producing bone lesions.

It is of utmost importance in older individuals to establish the presence or the absence of Paget's disease of bone and tumors of any part of the body either past or present. Metastatic bone lesions may occur many years after the removal of carcinoma of the breast or the thyroid gland. One case exhibited metastasis to the head of the humerus eight years after the removal of a carcinoma of the breast by radical mastectomy.

SITE OF TUMOR

Knowledge of the predilection of tumors of bone is a valuable aid in making a diagnosis. Osteogenic sarcoma is encountered more frequently in the humerus than any

of the other bones of the shoulder girdle. Solitary bone cysts are observed most frequently in the diaphysis close to the metaphysis; giant-cell tumors in the epiphyseal region (usually after ossification of the epiphyseal plate). The upper end of the humerus is a common site for chondrosarcomas. Ewing's tumors most frequently involve the shafts of long bones, while osteogenic sarcomas involve the ends of the shafts, the metaphyseal region (Fig. 290).

OBJECTIVE FINDINGS

A careful physical examination in most instances discloses pertinent clinical features upon which a presumptive diagnosis can be made. The skin over a neoplasm usually exhibits numerous dilated venous channels; its color is distinctive, being a reddish, dusky hue. In moderate and far advanced chondrosarcomas or osteogenic sarcomas in



FIG. 291 Enlargement of right extremity resulting from obstruction of venous and lymphatic circulation by hemangiosarcoma of the right scapula. The patient died of generalized metastasis (T A Shallow Jefferson Medical College.)

volving the bones of the shoulder girdle the characteristic configuration of the point of the shoulder is lost the bony landmarks being obscured by the tumor mass and the surrounding soft tissue reaction

Palpation of the tissues around the lesion often discloses increased local temperature especially in rapidly growing highly vascular lesions Unless the overlying tissue is involved the skin is freely movable over the tumor mass. The skin may be thin and stretched tightly over the mass occasionally ulceration occurs and the tumor may fungate through the skin This seldom occurs in bone sarcomas, unless an ill performed biopsy has been done it is observed more commonly in carcinomas

The size and the shape of tumors in the region of the shoulder as in other parts of the body vary greatly Chondrosarcomas are usually the largest tumors affecting this region Generally tumors arising in the

medullary and cortical portions of bone encounter greater resistance to growth and expansion than those arising from the surface of bone or beneath the periosteum, the latter group are faster-growing lesions and reach greater dimensions Interference with the venous and lymphatic circulation may result in pronounced enlargement of the extremity distal to the lesion (Figs 291 and 292) The distortion may approach such proportions as to destroy completely the functional usefulness of the arm. However size does not indicate the malignant or benign character of a tumor It is generally recognized that many small tumors may not be detected until after there is evidence of metastasis

PULSATING TUMORS

Highly vascular tumors may pulsate. However pulsation is a relatively rare feature of bone tumors, observed in lesions involving the ends of long bones and (less frequently) in lesions of the shafts. Osteolytic metastatic lesions from renal and thyroid cancer may produce a pulsating tumor Codman was of the opinion that pulsation of a tumor mass destroys the surrounding bone and causes expansion of the remaining thin bony shell Extension of the tumor down the medullary canal is restrained by the pulsating bone marrow

The number of pulsating giant-cell tumors reported in the literature is exceedingly small Except for the greater rapidity of growth and greater tendency toward recurrence after therapy either by surgery or irradiation these lesions in no way differ histologically from nonpulsating giant-cell tumors

BODY REACTION

As previously indicated a malignant osteogenic neoplasm may be ushered in with all the clinical manifestations of an acute infection. It is common knowledge that many cases of Ewing's tumor have been subjected erroneously to operative procedures under the diagnosis of acute osteomyelitis.

Roentgenographic study may fail to disclose the true nature of the lesion.

Because of the associated intense pain and the characteristic roentgenographic feature of localized increased bone density simulating a localized bone abscess, osteoid osteoma also has been operated upon under such mistaken diagnoses as Brodie's abscess and Garre's disease. One instance of an osteoid osteoma was curetted at one operation and drilled at another before the true nature of the disease was suspected. The symptoms were relieved only after the third operation, at which time a block resection was done on the bone which contained the nidus. To date, no osteoid osteoma of the upper one third or head of the humerus has been reported. Ten cases with involvement of the shaft of the humerus distal to its upper third are on record.

As a rule the general health of a patient with sarcoma is better than in carcinoma except in the terminal phases. In carcinoma there is always demonstrable anemia of varying degrees which becomes more marked as the tumor progresses. Many of the patients become extremely apprehensive

and irritable as a result of the severe pain which often interferes with their sleep.

ROENTGENOGRAPHIC STUDIES

Roentgenographic examination is even more important than physical examination and second only in importance to histologic study of bone lesions, in arriving at a diagnosis. In many instances carefully taken roentgenograms in anteroposterior, lateral and oblique views may give sufficient information to make a correct diagnosis. It must be pointed out that numerous and diverse interpretations may be derived from different roentgenologists. However, one well versed in the characteristic features of bone lesions can make a correct diagnosis in a considerable number of cases and a tentative one in others.

Coffman was cognizant of the importance of radiographic studies about the shoulder when he stated:

Bone sarcoma is practically the only fatal lesion which arises in the shoulder. We must learn to recognize it and to cure it. It should be our first thought in the case of a young patient complaining of a dull aching pain if any thickening of the bone were palpable or



FIG. 292 Cystic degeneration occurring in the carpal and metacarpal bones of an individual with extensive lymphedema of the extremity following intensive radiation of a primary bone tumor in the upper end of the humerus. (H. Ostrum, Philadelphia General Hospital.)

the pain persisted for more than a week we should seek help from the X ray

Exposures other than the routine conventional ones mentioned may be necessary to give more accurate and detailed information. Laminographic and stereoscopic exposures may add much pertinent information when more difficult diagnostic problems arise.

It should be emphasized that it is often impossible to establish a diagnosis by roentgenographic study, even though many bone lesions demonstrate roentgenographically characteristic features which should reveal their identity (this is especially true of benign lesions). Some tumors of bone for example give rise to bizarre roentgenograms which defy the most competent roentgenologist. It is interesting to note that many such lesions also usually exhibit histologic sections which fail to conform to any specific category. Many lesions mimic the features of others making a correct diagnosis by roentgenographic survey alone almost impossible.

Notable examples are syphilis of the bone which may be diagnosed erroneously as primary osteogenic sarcoma. Ewing's tumor is often mistaken for acute osteomyelitis and vice versa. Single lesions of metastatic carcinoma to bone may be interpreted as primary osteologic bone sarcoma. In some cases it is impossible to differentiate Ewing's tumor from metastatic bone lesions of neuroblastoma. Osteoid osteoma may resemble a bone abscess or Garré's disease and myositis ossificans has been mistaken for periosteal sarcoma of bone. Coley records an incidence of from 14 to 20 per cent error in roentgenographic diagnosis when checked by histologic examination.

The character of some bone lesions may point to generalized skeletal involvement which makes it imperative that a survey of the entire skeletal system be made. This is true of such lesions as multiple myeloma, parathyroid disease, fibrous dysplasia of bone, multiple exostosis and metastatic bone lesions. Roentgenographic study of the chest

is an essential part of a diagnostic investigation of bone tumors. Occasionally, the only evidence of the true nature of a bone lesion is the presence of metastatic nodules in the lungs. Repeated examination of the chest at regular intervals should be made to check the diagnosis of a bone lesion and to follow the course of its behavior, regardless of the choice of therapy instituted.

LABORATORY EXAMINATIONS

The role of laboratory investigations is progressively assuming greater proportions in the diagnosis of bone diseases. Valuable contributions have been made to this aspect of bone study by Albright, Reifstein, Woodard, Bodnansky, Gutman, Kutscher and many others.

The examinations which comprise a well rounded laboratory investigation are (1) complete blood count, (2) Wassermann reaction of blood and spinal fluid, (3) serum calcium level, (4) serum phosphorous level, (5) serum phosphatase level and (6) test for Bence Jones bodies in the urine. Other tests that may reveal information of value are sugar determinations, uric acid level which is elevated in gout, and blood proteins level which may be greatly elevated in multiple myeloma.

Alkaline phosphatase levels are normally high in growing individuals, and in normal reparative processes in which new bone is being formed such as in fractures. High values are also discernible in bone lesions characterized by new bone formation such as in osteogenic sarcoma, osteoplastic metastatic carcinoma to bone and Paget's osteitis deformans. Elevated phosphatase levels do not indicate any specific bone tumor or the malignant nature of bone lesions; it is simply an indicator of new bone formation in the course of a normal reparative process or by a neoplastic primary or secondary bone lesion.

In bone diseases exhibiting disturbance of calcium and phosphorous metabolism, the alkaline phosphatase values may be in-

creased, as in rickets, osteomalacia and hyperparathyroid disease.

Generally osteolytic lesions arising from carcinoma do not cause a rise in the serum alkaline phosphatase level. On the other hand in osteoplastic lesions, such as from cancer of the prostate gland, the level is invariably high. In a study made by Coley, it was observed that 75 per cent of all metastatic bone lesions from the prostate were osteoplastic and the remainder disclosed some new bone formation. Serum acid phosphatase is elevated in cases with metastatic bone lesions from prostatic cancer. Some authorities believe that a high serum acid phosphatase level is pathognomonic of cancer of the prostate.

HISTOLOGIC EXAMINATION

In the great majority of cases the diagnostic steps previously mentioned will yield sufficient information to make a correct diagnosis. But, in a small group, only a presumptive diagnosis can be arrived at. The last and final aid at our disposal to establish a diagnosis is histologic examination of the suspected tissue. Tissue may be obtained by surgical or aspiration biopsy.

Aspiration biopsy is preferred to surgical biopsy in many cancer clinics because of its simplicity and its relative high incidence of satisfactory results. While it is a method that should be adopted in all tumor clinics it is not without some disadvantages. Material obtained by aspiration may not be adequate to make a diagnosis. There is always the possibility of disseminating tumor cells along the path of the needle or of producing hemorrhage within the tumor, thereby favoring metastasis. The experience of Coley and Snyder with this method is significant. They recorded that in 568 aspiration biopsies of bone on 474 individuals there were no immediate complications and no evidence suggesting that the procedure had caused more rapid development of metastasis. Also in 67.5 per cent of 385 cases of primary and metastatic bone neoplasms a specific diagnosis was established by aspiration biopsy.

82 per cent of these cases yielded sufficient tissue to permit a diagnosis. In 14.5 per cent the tissue was not identified and in 1 case a benign lesion was diagnosed as a malignant tumor.

Aspiration of the iliac crest for bone marrow is gradually displacing sternal puncture formerly a valuable diagnostic procedure in cases of metastatic carcinoma, multiple myeloma and leukemia. Erl is of the opinion that marrow from the iliac crest permits an earlier diagnosis in the above lesions and gives a higher incidence of specific diagnoses than sternal puncture.

Surgical biopsy is still the method of choice in some institutions. In some cases it is the only method whereby tissue can be obtained for histologic examination. It is a procedure which should not be taken lightly and should be performed only by surgeons well versed in surgical technique and in the handling of tissues. Whenever possible the operation should be performed under tourniquet after the desired tissue is obtained the wound is closed in layers. No incision in a suspected malignant tumor should ever be left open or drained. Closure as described above should be carried out even in the event that severe hemorrhage demands packing of the wound. By so doing the chances of disseminating tumor cells and fungation of the tissues are minimized. In spite of the greater hazards of surgical biopsy, it yields more material for examination and a greater incidence of specific diagnoses than the aspiration method. It always should be performed when any radical surgical procedure is contemplated.

GIANT CELL TUMOR

INTRODUCTION

Giant-cell tumor is now definitely established as a benign disease of the bone primarily affecting the ends of the shaft of long bones. Many limbs were sacrificed needlessly until its true nature was recognized. Credit goes to Cooper, Paget, Nilater later to Gross, Stewart, Coley, Bloodgood and

the pain persisted for more than a week, we should seek help from the X ray

Exposures other than the routine conventional ones mentioned may be necessary to give more accurate and detailed information. Laminographic and stereoscopic exposures may add much pertinent information when more difficult diagnostic problems arise.

It should be emphasized that it is often impossible to establish a diagnosis by roentgenographic study, even though many bone lesions demonstrate roentgenographically characteristic features which should reveal their identity (this is especially true of benign lesions). Some tumors of bone, for example, give rise to bizarre roentgenograms which defy the most competent roentgenologist. It is interesting to note that many such lesions also usually exhibit histologic sections which fail to conform to any specific category. Many lesions mimic the features of others, making a correct diagnosis by roentgenographic survey alone almost impossible.

Notable examples are syphilis of the bone which may be diagnosed erroneously as primary osteogenic sarcoma. Ewing's tumor is often mistaken for acute osteomyelitis and vice versa. Single lesions of metastatic carcinoma to bone may be interpreted as primary osteologic bone sarcoma. In some cases it is impossible to differentiate Ewing's tumor from metastatic bone lesions of neuroblastoma. Osteoid osteoma may resemble a bone abscess or Carré's disease, and myositis ossificans has been mistaken for periosteal sarcoma of bone. Coley records an incidence of from 14 to 20 per cent error in roentgenographic diagnosis when checked by histologic examination.

The character of some bone lesions may point to generalized skeletal involvement which makes it imperative that a survey of the entire skeletal system be made. This is true of such lesions as multiple myeloma, parathyroid disease, fibrous dysplasia of bone, multiple exostosis and metastatic bone lesions. Roentgenographic study of the chest

is an essential part of a diagnostic investigation of bone tumors. Occasionally, the only evidence of the true nature of a bone lesion is the presence of metastatic nodules in the lungs. Repeated examination of the chest at regular intervals should be made to check the diagnosis of a bone lesion and to follow the course of its behavior, regardless of the choice of therapy instituted.

LABORATORY EXAMINATIONS

The role of laboratory investigations is progressively assuming greater proportions in the diagnosis of bone diseases. Valuable contributions have been made to this aspect of bone study by Albright, Reifstein, Woodard, Bodansky, Gutman, Kutscher and many others.

The examinations which comprise a well rounded laboratory investigation are (1) complete blood count, (2) Wassermann reaction of blood and spinal fluid, (3) serum calcium level, (4) serum phosphorous level, (5) serum phosphatase level and (6) test for Bence Jones bodies in the urine. Other tests that may reveal information of value are sugar determinations, uric acid level which is elevated in gout, and blood proteins level which may be greatly elevated in multiple myeloma.

Alkaline phosphatase levels are normally high in growing individuals and in normal reparative processes in which new bone is being formed, such as in fractures. High values are also discernible in bone lesions characterized by new bone formation, such as in osteogenic sarcoma, osteoplastic metastatic carcinoma to bone and Paget's osteitis deformans. Elevated phosphatase levels do not indicate any specific bone tumor or the malignant nature of bone lesions; it is simply an indicator of new bone formation in the course of a normal reparative process or by a neoplastic primary or secondary bone lesion.

In bone diseases exhibiting disturbance of calcium and phosphorous metabolism, the alkaline phosphatase values may be in-

The traumatic theory is the one most generally accepted

Codman believed that rupture of a nutrient artery which fails to clot was the origin of all giant-cell tumors. From this nidus arises a pulsating mass of tissue which produces resorption of the adjacent bony cancellous trabeculae. The mass extends centrally because the cancellous bone is less resistant than cortical bone. Extension of the tumor into the shaft is prevented by the pulsating bone marrow.

Geschickter and Copeland contend that trauma following subperiosteal hemorrhage interrupts normal blood supply to the cortex of the adjacent bone. This results in increased osteoclastic activity in the subcortical region of the traumatized cortex and finally by hyperplasia of osteoclasts, a tissue designated giant-cell tumor is formed.

MACROSCOPIC PATHOLOGY

Macroscopic examination of early tumors discloses their subcortical origin. They are eccentrically located, tending to invade the cancellous bone centrally, while the more-resistant cortical bone acts as an effective barrier to peripheral expansion of the tumor. More advanced lesions reveal expansion of the epiphyseal region with marked thinning of the cortical shell which may be perforated in several places (Fig. 293). The periosteum tends to limit the tumor mass when the continuity of the cortex is broken; even this last barrier may be broken in very aggressive tumors, causing invasion of the surrounding soft tissue. The tumor rarely invades the joint cavity; the articular cartilage resists invasion. It is not uncommon to find the cartilage still intact sitting like a cap on a mass of tumor tissue (Fig. 293) even in the most advanced lesions, with total destruction of all bony elements. Extension into the shaft does not proceed too far beyond the metaphyseal region, apparently the pulsating bone marrow tends to limit the advance of the tumor. Fibrous tissue proliferation in this region also restrains the tumor from advancing into the shaft.

The tissue varies in color and consistency in different tumors. Usually it is friable, disintegrates readily, bleeds freely and ranges in color from red to black. Occasionally, it is firm and puttylike and of a grayish white color. Fibrous trabeculae—some coarse, others fine—are often encountered throughout the tumor, appearing to extend into the substance of the tumor from its periphery. At operation the tumor may be very vascular, as evidenced by marked generalized oozing and the presence of large, spurring vessels. Frank hemorrhage may be difficult to control in such lesions. Other tumors may be avascular, permitting curettage without difficulty.

MICROSCOPIC PATHOLOGY

The histologic picture varies in different tumors or in different areas of the same tumor, depending upon the stage of the neoplasia's development and the secondary changes present in the tumor tissues. In tumors exhibiting no (or only minimal) secondary alterations, the tumor tissue comprises a characteristic stroma made up of mononuclear spindle or ovoid cells, varying numbers of multinuclear giant cells and small amounts of intercellular collagenous substance consisting of fine fibers.

The ratio of spindle to ovoid cells varies in different tumors. These cells possess a single round or elongated nucleus which occupies a considerable portion of the cell. Each nucleus contains a well-defined central nucleolus and a moderate amount of evenly distributed chromatin material. As a rule, the ovoid cells are found in greater numbers than the spindle cells. The outline of the cytoplasm of the stromal cells may be indistinct and exhibit delicate cytoplasmic processes. Jaffe records that the most plausible origin of stroma cells is from mesenchymelike supporting connective tissue of the marrow.

Irregularly distributed throughout the stroma are the multinucleated giant cells, measuring from 30 to 100 or more microns. The number of giant cells in each field



FIG 293 Giant-cell tumor involving the entire upper end of the shaft of the humerus in a female aged 23. Note the expansile nature of the tumor; only a thin cortical shell remains. No bony shell is visible on the inner aspect of the tumor. At operation it was noted that the tumor was limited in this region by the thickened periosteum only. Observe the intact cartilage on the superior aspect of the head.

others for recognizing and emphasizing the benignity of this neoplasm. However it is now known that giant-cell tumors occasionally take on malignant characteristics and may metastasize to the lungs. Such malignant changes have been noted when tumors were first seen and before any form of therapy had been instituted. The majority of malignant giant-cell tumors have been noted following some form of therapy usually irradiation.

AGE AND SEX

Giant-cell tumors are rarely found in young children or in individuals past mid

dle life, the highest incidence in any series recorded is in the third decennium. Geschickter and Copeland report that 40 per cent of all giant-cell tumors occur in the third decade. Coley records 48 out of 124 cases occurring between the ages of 21 and 30 years, 78 in 124 being between the ages of 21 and 40 years.

There appears to be no predilection as to sex. Males and females are affected about equally.

SITE

The proximal end of the humerus ranks fourth in frequency. More common sites in order of frequency are the distal end of the femur, the proximal end of the tibia and the distal end of the radius.

Although giant-cell tumors are primarily solitary neoplasms of the cancellous portions of tubular bones, they occur in all bones of the body, including the skull. Giant-cell tumors usually begin in the subcortical portion of the epiphysis and by gradual expansion and destruction of the less-resistant cancellous bone occupy a more central position. Occasionally the tumors are completely eccentric, appearing to develop beneath the periosteum; such tumors are designated as subperiosteal giant-cell tumors. Subperiosteal giant-cell tumors have been reported as arising from the shaft of the long bones at a distance from the epiphyseal region. Some investigators are of the opinion that the latter group is a distinct entity, these tumors being sequelae of subperiosteal hematoma.

ETIOLOGY

Numerous theories have been advanced to explain the origin of giant-cell tumors. Barnes's theory of hemorrhagic osteomyelitis, disproved by Stewart (1922), has been discarded. There are still supporters of the neoplastic theory, some of whom postulate that the tumor arises from marrow cells; others believe that it originates from and consists of bone formative cells (Willis) which possess osteoclastic characteristics.

irregular whorled arrangement, demonstrating uniformly signs of atypism. These tumors are outright malignant neoplasms and usually metastasize to the lungs. They usually represent malignant transformations of Grades I and II but occasionally begin as Grade III tumors (Fig. 294)

CLINICAL FEATURES

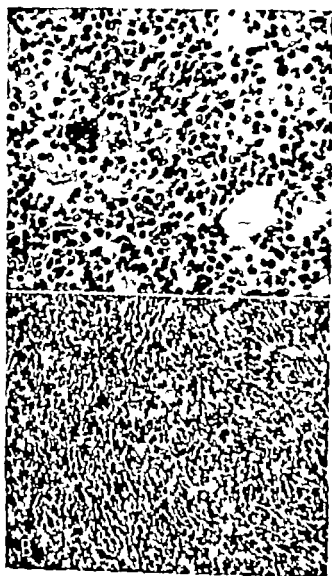
Giant-cell tumors are usually encountered in the third decade of life. Usually a definite history of trauma to the shoulder can be elicited. Pain of varying degrees is the cardinal symptom. As a rule the pain is not severe, may be intermittent and is often referred to the insertion of the deltoid muscle

or the posterior region of the shoulder. At first the pain is often described as rheumatic in nature. It is accentuated by activity and relieved by rest. Motion at the shoulder joint in the early phases of the disease may not be impaired materially.

Pain becomes more severe and constant as the tumor progresses. The patient holds the arm to the side and protects it against unguarded movements. Contracture and shortening of the adductor muscles follow, producing greater restriction of motion. No case under the author's personal observation involved a completely frozen shoulder; however, motion is usually possible only below the horizontal. Pain interferes with

FIG. 294B (Top) Tissue from a giant-cell tumor already classified as belonging to Grade II ($\times 285$). The lesion occupied the lower end of a femur in a man of 36. There was no preoperative irradiation of the tumor. Note the compaction of the stromal cells and the presence of multiple nuclei in some of them. More of the stromal cells showed mitoses than show them in a tumor of Grade I. Despite postoperative irradiation the lesion progressed and two and one-fourth years later the histologic picture had changed completely; the lesion having become frankly malignant.

(Bottom) Tissue from a giant-cell tumor of Grade II that occupied the lower end of a femur in a 23-year-old woman ($\times 90$). The tissue shown represents material obtained from the lesion through curettage some time after a thorough course of radiation therapy had failed to control its progression. Subsequently the lesion became infected and fungated and the limb was amputated. Note that the field illustrated contains numerous densely compacted spindle-shaped stromal cells but that hyalinization of the stroma, which would indicate an irradiation effect, is absent. Note also that hardly any giant cells are to be seen in the photomicrograph. In other fields of the tumor however they were very numerous and the histologic architecture altogether so characteristic that there could be no doubt that one was dealing with a giant-cell tumor in spite of the concomitant presence of more ominous looking areas like the one shown (Jaffe et al. Arch. Path. 30: 1004).



varies, generally, the number demonstrable in the low-power field ranges from 10 to 35 cells. The nuclei of typical giant cells generally occupy a central position in the cell, their number varying from 15 to 100 or more in each cell. They display a remarkably close resemblance to the nuclei of ovoid stromal cells. Some workers are of the opinion that giant cells are formed by fusion of the stromal cells.

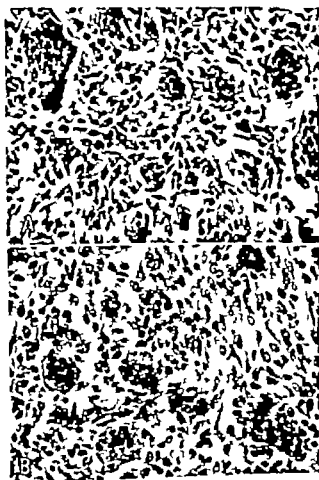
Collagen fibers are not abundant in uncomplicated giant-cell tumors. When the stromal cells exhibit a loosely arranged pattern, the collagenous material is more abundant than in a compact arrangement of the stromal cells. Vascularity of the tumor tissue is also a variable feature; the vascular spaces are thin walled, composed of flattened endothelial cells. Tumor cells are frequently found in close relation with the vessel walls.

Areas of hemorrhage are frequently encountered scattered throughout the tumor.

Jaffe believes that a typical, uncomplicated giant-cell tumor discloses a characteristic stromal pattern. Change in this stroma indicates the aggressiveness of the tumor, certain features pointing to a frankly malignant giant-cell tumor. He describes three grades of aggressiveness.

Grade I is the least aggressive. In this group there is a loose arrangement of the stromal cells with no appreciable atypism of the stroma cells. Grade II is more aggressive. Tumors in this category tend to recur and finally may assume the characteristics of Group III. The stroma cells are in greater abundance than in Group I, forming a compact stromal pattern and disclosing definite manifestations of atypism. Grade III is characterized by a dense compact stroma with an

FIG 294A. (Top) Tissue from a giant-cell tumor of Grade I that occupied the head and neck of a humerus in a woman of 30 ($\times 275$). There was no preoperative irradiation of this tumor. Note the abundance of large giant cells and the predominantly spindle-shaped and not closely compacted stromal cells between them. The stromal cells presented few mitoses and no evidences of atypism. Altogether this histologic picture represents what can definitely be called a 'benign' giant-cell tumor and indeed curettage and postoperative irradiation of the lesion were followed within a year by substantial repair of the affected area.



(Bottom) Tissue from a giant-cell tumor still properly classifiable as belonging to Grade I but no longer so clearly benign as the tumor above ($\times 245$). The lesion occupied the head and neck of a femur in a woman of 22. There was no preoperative irradiation of the tumor. Note that the giant cells are rather abundant between the stromal cells and that the latter are abundant but not closely compacted. The stromal cells do not show appreciable atypism but here and there some of them have large nuclei and an occasional one even has two nuclei. In this case, postoperative irradiation was not followed by prompt clinical improvement. Eventually we lost sight of this patient, as she left the country (Jaffe et al. Arch. Path. 30:1002).



FIG 295 Giant-cell tumor in female, aged 26. Observe pronounced expansion of the upper end of the humerus; the remaining cortical shell is thin on the outer aspect of the humerus and absent on the inner aspect. A cartilaginous cap still remains intact; the tumor is traversed by trabeculae of varying thicknesses giving the tumor mass a bubble-like appearance.

served in the early states. Later, with resorption of the cancellous bone, the lesion occupies a more central position; fine or coarse trabeculae may traverse the tumor, giving it a bubblelike pattern. The upper end of the humerus expands; its cortical shell becomes thin and it may even show evidence of perforation by a break in its continuity. Generally, the tumor does not extend far beyond the limits of the metaphysis into the shaft. Advanced lesions exhibit total destruction of all the bony elements in this region; the limits of the tumor being maintained by a barely perceptible thin bony shell and a thickened periosteal sheath. Occasionally only the periosteal tube remains in certain areas around the tumor. The cartilage resists joint invasion

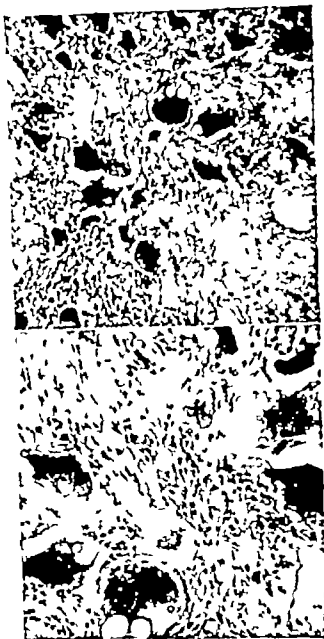


FIG 296 Tissue obtained from giant cell tumor depicted in Figure 295. The stroma comprises mononuclear spindle and ovoid cells and small amounts of intercellular collagenous substances consisting of fine fibers. Many multinucleated giant cells of varying sizes are distributed irregularly throughout the stroma. [Photomicrographs ($\times 160$ and $\times 320$)]

and becomes calcified and new bone may form on its under surface so that in the roentgenograms it is discernible as a thin smooth line of increased density (Fig 295). Characteristic of this lesion at no time is there radiographic evidence of periosteal reaction, soft tissue mass or swelling. In the event of fracture, displacement of the fragments is not pronounced. No evidence of

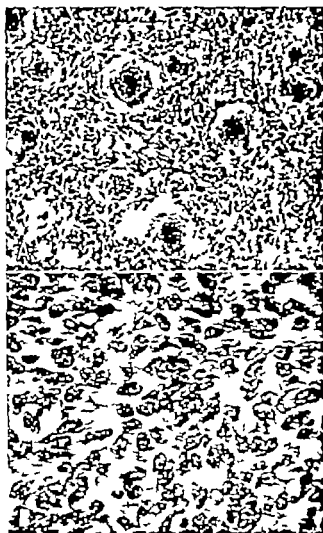


FIG 294C (Top) Tissue from a giant cell tumor of Grade II which occupied the lower end of a femur of a 44-year old man ($\times 150$). Radiation was not given preoperatively but was used after thorough curettage. Nevertheless there was a recurrence associated with infection and fungation necessitating ablation of the limb about one and a half years later. The field illustrated is from the tissue originally curetted out. Note between the giant cells, the densely compacted stromal cells in a somewhat irregular whorled arrangement. In the field illustrated these cells, when studied in detail did not present much atypism but in other fields (below) they did. This shows the necessity for studying details of the stromal cells from many areas prognosing the lesion by its most ominous-looking parts.

(Bottom) Stromal cell field showing atypism from same tumor ($\times 550$). Several of the cells contain two or more nuclei; the nuclei in general are large and many show hyperchromatism (Jaffe et al. Arch. Path. 30 1006).

the patient's sleep and becomes excruciating if the individual lies on the affected shoulder. Occasionally, swelling of the extremity distal to the lesion is demonstrable. Spontaneous fracture may occur, all symptoms now become more pronounced and functional impairment of the extremity is increased.

PHYSICAL FINDINGS

During the early stages of the disease no physical evidence of a tumor may be apparent. Impairment of normal movements at the shoulder joint is manifest early and increases with progression of the tumor. Except in very thin individuals swelling is not readily visible or palpable because the head of the humerus is almost completely covered with the fleshy deltoid muscle. Pressure over the upper end of the humerus and over the tuberosity of the humeral head invariably elicits complaints of tenderness.

In the later stages with expansion of the upper end of the humerus the affected shoulder appears fuller and more prominent when compared with the normal side. Palpation reveals a smooth, symmetric enlargement of the upper end of the humerus. All movements except within a small range, are painful. Pressure over the distended portion of the bone shows considerable tenderness to be present. The skin and the muscle are freely movable over the involved bone and exhibit no significant diagnostic features. Pulsation is an exceedingly rare sign but it may occur. Up to 1946 only seven cases of pulsating giant-cell tumors were reported in the literature, this feature being more commonly associated with thyroid or renal metastasis to bone.

ROENTGENOLOGIC FEATURES

Certain characteristic radiologic features of a giant-cell tumor plus the fact that it occurs in the epiphyseal region of the humerus in a specific age period (usually the third decade) render roentgenographic interpretation of the lesion relatively simple. Only an eccentrically placed radiolucent area in the epiphyseal region may be ob-

stroma of the tumor. Clinical and roentgenologic features are not sufficient to establish a positive diagnosis. Giant cell tumors which undergo malignant transformation following irradiation, surgery, or both manifest their true character by recurrence of symptoms at varying periods following the initial therapeutic measures. Microscopic study confirms the diagnosis. It is obvious, of course, that clinical evidence of pulmonary metastasis following the treatment of a supposedly benign giant-cell tumor confirms the malignant nature of the lesion.

Osteogenic Sarcoma. Medullary or central osteogenic sarcoma may arise occasionally in the proximal end of the humerus. The neoplasm may run a clinical course similar to that of giant-cell tumor. Roentgenographically a radiolucent lesion is demonstrable in the epiphyseal region consistent with an osteolytic process; no new bone formation may be discernible. Hence, even by roentgenographic study the malignant lesion may not differ essentially from giant-cell tumor. Histologic examination is the only diagnostic aid which will establish the identity of the lesion.

Epiphyseal Chondromatous Giant Cell Tumor. This rare benign lesion first described by Codman (1931) is located in the greater tuberosity of the head of the humerus. It occurs before ossification of the epiphyseal plate in a younger age group than giant-cell tumors. The tumor does not extend beyond the epiphyseal cartilage of the articular head. No trabeculations are discernible in the substance of the tumor which has a "cotton wool" appearance in the roentgenogram. These tumors run a mild clinical course; microscopic examination identifies them readily and reveals that a considerable portion of these tumors consist of cartilage.

Hyperparathyroidism (Generalized Osteitis Fibrosa Cystica). Osseous lesions associated with hyperparathyroidism are nonneoplastic and multiple, involving many of or all the bones of the body. The lesion is more-or-less diffuse, chiefly affecting the shaft of the bones. Microscopic examination



FIG 298 Giant-cell tumor depicted in Figure 295 4 months after curettage and packing of the remaining cavity with bone chips from the iliac crest and rib bone from the bone bank, used as struts to prevent collapse of the cavity. Observe that the rib struts are serving their function, and the bone chips are coalescing.

may fail to differentiate the lesion from giant-cell tumor because of the close similarity in the histologic sections.

Diagnosis is made readily by blood studies. Normal serum levels of calcium, phosphorus, and phosphatase occur in giant cell tumor, while the serum calcium and phosphatase are elevated and the phosphorus is lowered in generalized osteitis fibrosa cystica.

Metastatic Carcinoma. Occasionally isolated metastatic bone lesions of the upper end of the humerus may occur, particularly from renal or thyroid cancer. Roentgenograms may reveal an osteolytic and even expansile lesion. The lesion is pictured as a radiolucent area in the bone with no evi-



FIG 297 Osteolytic metastatic breast carcinoma in upper end of the humerus resembling a giant-cell tumor in a female 42 years of age. (B P Widmann and J C Howell, Philadelphia General Hospital.)

repair is discernible clinically or radiographically.

Although the above features are more or less indicative of a giant-cell tumor the diagnosis can only be a presumptive one. Occasionally osteolytic metastatic lesions and central osteogenic sarcomas in the head of the humerus depict radiographic features closely resembling those described above (Fig 297).

DIAGNOSIS

The age of the patient and the location of the tumor are the two most significant factors in establishing the diagnosis of giant-cell tumors. Nevertheless there are several entities, neoplastic and nonneoplastic which may resemble closely the characteristics of these tumors both clinically and roentgenographically. It becomes apparent that before any therapeutic measures are instituted the diagnosis of giant-cell tumor should be confirmed first by histologic examination

Osseous lesions which are more apt to be confused with giant-cell tumor are (1) bone cyst, (2) malignant giant-cell tumor (3) medullary (central) osteogenic sarcoma (4) epiphyseal chondromatous giant-cell tumor (Codman's) (5) hyperparathyroidism and (6) metastatic carcinoma.

Bone Cyst (Solitary Osteitis Fibrosa)
The features of bone cysts which resemble giant-cell tumors are usual location in the upper end of the humerus close to the epiphyseal plate expansive tendencies (the transverse dimensions of the shaft increase and the cortical shell may become so thin that in many instances fracture ensues), histologic structure and benign nature.

Giant-cell tumors may be differentiated from bone cyst by their occurrence in an older age period after epiphyseal ossification is completed. While giant-cell tumors are located in the region of the epiphysis proximal to the epiphyseal plate, bone cysts occur in individuals of an earlier age group in whom the epiphyseal line is still open the lesion arising on the diaphyseal side of the metaphysis.

The clinical course of bone cysts differs from that of giant-cell tumors in their mildness. Many bone cysts cause no symptoms and are discovered only accidentally or when a fracture of the humeral shaft occurs. Following fracture there is evidence of an active reparative process which may not only heal the fracture but may obliterate the cyst. Such a reparative process never has been observed following pathologic fracture in giant-cell tumors.

Histologic examination may be very confusing because of the close resemblance between giant-cell tumors and bone cysts. The diagnosis depends chiefly upon the clinical course of the lesions, the age of the patient, the location of the lesion in reference to the epiphyseal line and the roentgenographic features.

Malignant Giant Cell Tumors. The malignant nature of a giant-cell tumor can be definitely detected only by microscopic examination which discloses the sarcomatous

and surgery. However, there is much disagreement as to which is the better method. Some investigators contend that treatment by roentgen rays causes less impairment of joint function and is followed by fewer recurrences. On the other hand, investigators favoring surgery maintain that it is followed by shorter periods of disability, that fracture is a common sequela of irradiation, and that sarcoma may arise in irradiated bone. Coley notes that 25 per cent of the cases of giant-cell tumors of long bones treated by roentgen rays sustained pathologic fractures.

In general most workers favor the surgical approach to this problem and employ irradiation only in tumors inaccessible to surgery, or those in which the neoplasm has advanced to a stage that complete surgical extirpation of the tumor tissue is impossible, or in cases in which there has been total disruption of the joint with invasion of the tumor tissue into the joint cavity.

The surgical procedures employed are curettage, resection and amputation.

Curettage. Giant-cell tumors in the upper end of the humerus are most amenable to curettage. Advanced lesions located in the humerus (Fig. 298) can be treated by curettage if they are present in weight bearing bones; they are not acceptable for surgery because of subsequent severe impairment of joint function.

TECHNIC OF CURETTAGE. Adequate visualization of all aspects of the upper end of the humerus is essential to evaluate correctly the extent of the lesion and to gain access to all tumor tissue.

The incision of Cubbins et al. affords such an exposure (see Chap. 11). The skin incision starts along the anterior border of the deltoid. It is continued laterally on the inferior border of the clavicle, around the acromion process and along the inferior margin of the spine of the scapula.

After complete hemostasis of the skin and the subcutaneous tissue the skin edges are fastened to towels by skin or towel clips to protect the wound edges from exposure

and contamination. The interval between the deltoid and the pectoralis major is developed proximally to the lower border of the clavicle, taking care to avoid injury to the cephalic vein which is displaced medially with the edge of the pectoralis major muscle. Next the deltoid muscle is reflected subperiosteally from the clavicle, the acromion and the spine of the scapula. Gently the entire muscle mass is displaced downward, thereby exposing the superior, anterior, lateral and posterior aspects of the glenohumeral joint.

At this point emphasis should be placed on the care and the gentleness to be exercised while obtaining the above exposure. The soft tissues are covered and packed away from the involved bone by large hot sponges. This will prevent contamination of the tissues by tumor material as it is being removed from the bone cavity.

As a rule, the periosteal tube around the affected area of the humerus is intact. It is divided longitudinally over the lateral aspect of the greater tuberosity and reflected to either side. The exposed thin cortical shell is perforated readily with a bone gauge and the opening is enlarged by rongeurs until all the areas within the cavity are accessible. The tumor tissue is removed with a large curette, taking care to avoid spilling. An angular curette facilitates its removal from the roof of the cavity. Curettage is continued until normal healthy bone is reached. The cavity is then flushed with normal saline solution and sucked dry. This is done repeatedly until all loose tissue particles are removed. The interior of the cavity is swabbed with a solution of saturated zinc chloride and then the cavity is flushed again with normal saline solution. Next, the cavity is packed tightly with a hot gauze sponge, left in situ while bone chips are being prepared to fill the defect.

If the cortical shell of the humerus is intact and the defect is small, the cavity is packed with small bone chips obtained from either or both the iliac crest and refriger-

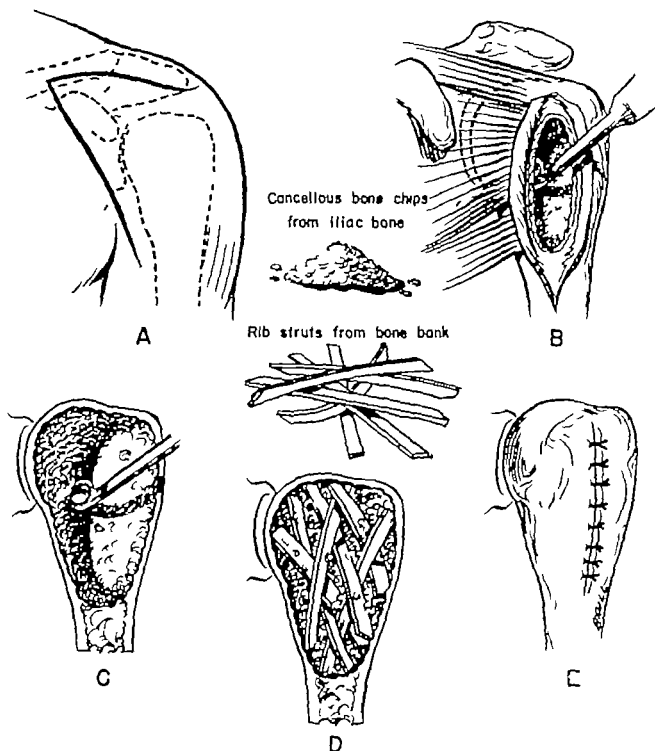


FIG. 299 Technic of curettage of giant-cell tumor and packing cavity with bone chips and bone struts.

dence of new bone formation. Diagnosis is made in accordance with the age of the patient (such lesions usually occur after the third decade), the general condition, the demonstration of a primary lesion and microscopic examination of the tissue.

Other osseous lesions that must be considered in a differential diagnosis are cen-

tral chondroma and such tumors of the marrow cells as plasma-cell myeloma, myelocytoma, lymphocytoma and erythroblastoma.

TREATMENT

Essentially there are two methods of treatment for giant-cell tumors: irradiation

EPIPHYSAL CHONDROMATOUS GIANT CELL TUMOR (CODMAN) [BENIGN CHONDROBLASTOMA OF BONE (JAFFE)]

INTRODUCTION

In 1931 Codman reported nine cases of this tumor which he found in the *Sarcoma Registry*. They passed under such erroneous diagnoses as osteogenic sarcoma, chondrosarcoma and giant-cell tumors. He believed these tumors were found only in the region of the humeral head and that they were atypical giant-cell tumors. He wrote,

It is my belief that the peculiar structure of the head of the humerus makes the picture of giant-cell tumor so modified that typical cases do not often occur.

Since the first description of these lesions, they have been observed in the epiphyseal regions of other long bones (lower end of the femur and upper and lower ends of the tibia).

ORIGIN

Investigators differ as to the origin of epiphyseal chondromatous giant cell tumors. Codman was of the opinion that this type of neoplasm was a benign giant-cell tumor arising in the region of the tuberosities of the head of the humerus at an age before complete ossification of the epiphyseal cartilages; that the peculiar epithelioid cells which merge into a low grade type of cartilage cell found in the tumor arose from the epiphyseal cartilage. Willis reports a case whose histologic structure supports the views of Codman.

On the other hand, Geschickter describes this lesion under the title of "chondroblastic tumors, benign and malignant," believing that these lesions arise from the metaphyseal side of the epiphyseal line and that the proliferating tissue was chondroblastic rather than composed of giant cells. Coley states that he knows of no verified instance of metastasis of this tumor and that in all the cases under his observation a cure was effected by conservative treatment. Jaffe

and Lichtenstein (1942) convinced that the tumors have a cartilaginous origin name the lesion benign chondroblastoma of bone. Despite the inconsistencies as to its origin, it is generally conceded that this tumor is a definite entity possessing benign characteristics.

CLINICAL FEATURES

A survey of the cases reported in the literature discloses that the tumor occurs at a period in life when the epiphyseal plates of the upper end of the humerus are still open. Codman's nine cases ranged from 12 to 24 years of age. Males are affected more frequently than females. As previously mentioned the region of the tuberosities of the upper end of the humerus primarily the greater tuberosity is involved most commonly. The onset and the clinical course are mild.

In Codman's series the duration of the disease before treatment varied from 5 to 36 months. Trauma as an etiologic factor has not been proved although it may make the patient cognizant of an existing lesion. Pain and restriction of shoulder motion are the cardinal symptoms. Later swelling of the upper end of the humerus may be demonstrable also tenderness over the greater tuberosities may be elicited by firm pressure over this region. The benign nature of the neoplasm is evidenced by the fact that conservative measures result in a permanent cure.

ROENTGENOGRAPHIC FEATURES

Benign chondroblastomas usually occupy an eccentric position in the greater tuberosity. No reported tumor ever has extended beyond the proximal epiphyseal line to reach the articular cartilage of the head of the humerus except in one case reported by Jaffe. These tumors present a fluffy cotton wool look, are relatively small and are sharply demarcated from the surrounding normal bone by an area of increased density. Irregular areas of calcification scattered through the tumor may give it a mul-

ated bone bank sources. Occasionally, after curettage the resulting defect is large, or the cavity collapses, after its contents have been removed as the result of dissolution of the cortical shell. In these instances, it is advantageous to use 4 or 5 long strips of bone in the cavity as struts—placed parallel with the long axis of the shaft of the humerus—and to pack the spaces between the struts with tiny cancellous bone chips. Rib bones split in halves make admirable bone struts for the purpose. (A supply of rib bones is always available in the bone bank.)

The incised periosteum is closed over the defect with interrupted cotton sutures, and the deltoid is reattached to its normal position. Several interrupted sutures close the interval between the deltoid and the pectoralis major muscles. Finally, the skin edges are carefully approximated.

Small defects in the humerus need not be supported by any type of external fixation. A bandage applied firmly over the wound together with a sling, affords sufficient protection to the extremity. In fact, early mobilization of the shoulder joint should be encouraged. This prevents the formation of adhesions between the soft tissue strata and contracture of the adductor muscles.

In the event of a pathologic fracture prior to curettage or a large defect with the cortical shell so thin that bone fracture is inevitable if unprotected following curettage the part should be protected until new bone formation obliterates the defect. The best method of external fixation is a shoulder spica made of plaster-of-paris applied immediately after operation.

Resection. This procedure is indicated only rarely for giant-cell tumors in the upper end of the humerus because of the subsequent impairment of shoulder joint function. Resection is justifiable in this region only if there is recurrence of the tumor after curettage. Some shoulder joint function may be restored following resection of the upper end of the humerus by replacing the resected bone with the proximal end of

the fibula (Fig 357, top right and bottom).

Amputation. Better understanding of the nature and the cause of giant-cell tumors condemns amputation of the upper extremity as primary treatment of these lesions. Amputation is indicated only when there is clinical and histologic evidence that the neoplasm has undergone malignant transformation (for technic see Chap 11) following treatment by irradiation or surgery. Occasionally such undesirable sequelae as infection and irradiation necrosis may render the extremity useless; here amputation is advisable. Primary amputation is performed however, only when histologic study reveals frankly the existence of a malignant giant-cell tumor.

PROGNOSIS

Many factors must be evaluated when considering the prognosis of giant-cell tumors. Notably are the age of the patient, the site of the tumor and characteristics of the stromal cells. In general the outlook is better in young individuals and lesions in the upper extremity give a more favorable prognosis than those in the lower extremity (Coley). The work of Jaffe on aggressiveness of giant-cell tumors based on changes in their stromal cells provides some aid in prognosticating the clinical behavior of a given tumor.

Finally the prognosis is unfavorable in lesions which tend to recur following treatment. Such behavior should arouse suspicion of malignant transformation and should influence the prognosis accordingly.

PREOPERATIVE AND POSTOPERATIVE RADIATION

There is general agreement that preoperative irradiation is not desirable; mild dosage has little effect on the course of the tumor and heavy radiation may delay or inhibit new bone formation. Postoperative radiation may be harmful for the same reasons and is needless therapy if thorough curettage has been performed.

EPIPHYSEAL CHONDROMATOUS
GIANT CELL TUMOR (CODMAN)
[BENIGN CHONDROBLASTOMA
OF BONE (JAFFE)]

INTRODUCTION

In 1931 Codman reported nine cases of this tumor which he found in the *Sarcoma Registry*. They paraded under such erroneous diagnoses as osteogenic sarcoma, chondrosarcoma and giant-cell tumors. He believed these tumors were found only in the region of the humeral head and that they were atypical giant-cell tumors. He wrote,

It is my belief that the peculiar structure of the head of the humerus makes the picture of giant-cell tumor so modified that typical cases do not often occur.

Since the first description of these lesions, they have been observed in the epiphyseal regions of other long bones (lower end of the femur and upper and lower ends of the tibia).

ORIGIN

Investigators differ as to the origin of epiphyseal chondromatous giant-cell tumors. Codman was of the opinion that this type of neoplasm was a benign giant-cell tumor arising in the region of the tuberosities of the head of the humerus at an age before complete ossification of the epiphyseal cartilages that the peculiar epitheloid cells which merge into a low grade type of cartilage cell found in the tumor arose from the epiphyseal cartilage. Willis reports a case whose histologic structure supports the views of Codman.

On the other hand Geschickter describes this lesion under the title of "chondroblastic tumors benign and malignant" believing that these lesions arise from the metaphyseal side of the epiphyseal line and that 'the proliferating tissue was chondroblastic rather than composed of giant cells'. Coley states that he knows of no verified instance of metastasis of this tumor and that in all the cases under his observation a cure was effected by conservative treatment. Jaffe

and Lichtenstein (1942) convinced that the tumors have a cartilaginous origin name the lesion 'benign chondroblastoma of bone'. Despite the inconsistencies as to its origin, it is generally conceded that this tumor is a definite entity possessing benign characteristics.

CLINICAL FEATURES

A survey of the cases reported in the literature discloses that the tumor occurs at a period in life when the epiphyseal plates of the upper end of the humerus are still open. Codman's nine cases ranged from 12 to 24 years of age. Males are affected more frequently than females. As previously mentioned, the region of the tuberosities of the upper end of the humerus primarily the greater tuberosity is involved most commonly. The onset and the clinical course are mild.

In Codman's series the duration of the disease before treatment varied from 5 to 36 months. Trauma as an etiologic factor has not been proved, although it may make the patient cognizant of an existing lesion. Pain and restriction of shoulder motion are the cardinal symptoms. Later swelling of the upper end of the humerus may be demonstrable also tenderness over the greater tuberosities may be elicited by firm pressure over this region. The benign nature of the neoplasm is evidenced by the fact that conservative measures result in a permanent cure.

ROENTGENOGRAPHIC FEATURES

Benign chondroblastomas usually occupy an eccentric position in the greater tuberosity. No reported tumor ever has extended beyond the proximal epiphyseal line to reach the articular cartilage of the head of the humerus, except in one case reported by Jaffe. These tumors present a 'fluffy cotton wool look' are relatively small and are sharply demarcated from the surrounding normal bone by an area of increased density. Irregular areas of calcification scattered through the tumor may give it a mul-



FIG 300 Chondroblastoma of the humerus in a male aged 23. Observe the mottled appearance resulting from irregular areas of calcification within the cartilaginous tumor. Also observe the pronounced increase in cortical bone in the shaft of the humerus immediately below the tumor, a characteristic feature of this tumor.

tilocular, flocculent appearance. Trabeculations such as seen in giant-cell tumors are absent. Unlike giant-cell tumors, new bone is formed on the outer surface of the cortex of the shaft immediately below the tumor. This periosteal response is more evident in the older lesions (Fig. 300).

MICROSCOPIC FEATURES

Histologic study reveals that benign chondroblastoma consists essentially of masses of immature cartilage cells embedded in a cartilaginous matrix. Occasionally, areas of calcification and ossification are found in the matrix. Varying numbers of giant cells are scattered throughout the tumor.

DIAGNOSIS

As previously noted, this lesion has often been mistaken for osteogenic sarcoma, chondroblastic sarcoma, and chondrosarcoma. The location of the tumor, its benign course, and long duration of symptoms together with its histologic features distinguish it from the aforementioned neoplasms.

TREATMENT

Radical surgical procedures never are indicated in these lesions. The treatment of

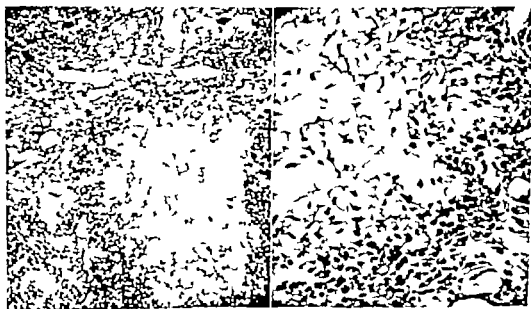


FIG 301 Benign chondroblastoma. The tumor tissue comprises stellate cells in a myxomatous stroma, fibroblastic proliferation, and numerous chondroblasts. (Photomicrographs $\times 125$ and $\times 250$.)

choice is careful curettage of the affected area or excision of the mass where possible. Surgical intervention is preferred to irradiation.

Prognosis

All the cases of Codman, Jaffe and Coley have responded to conservative treatment. Benign chondroblastomas do not tend to recur after adequate conservative treatment. As to eradication of the tumor and restoration of function at the shoulder joint, the prognosis is generally excellent.

SOLITARY BONE CYST

INTRODUCTION

Solitary bone cyst is a benign neoplasm whose clinical, roentgenographic and histologic characteristics have established it as a definite entity. It has a predilection for the ends of long bones, its most prevalent sites being the upper ends of the humerus and the femur. The close similarity of this lesion to giant-cell tumor has been the source of much controversy relative to its origin.

Three distinct varieties of this lesion are recognized: (1) The most common form is the bone cyst found in young children in the metaphysis of long bones (on the diaphyseal side of the epiphyseal plate). (2) The latent bone cyst observed in older children and adults is located in the shaft of the bone at some distance from the epiphyseal line (an older form of first variety which during the period of bone growth has migrated toward the middle of the shaft). (3) That group encountered during adolescence bordering on the epiphyseal disk and displaying more acute clinical manifestations than do the other two varieties. Histologically they bear close resemblance to giant-cell tumors. Coley refers to this classification as an intermediate form between a simple bone cyst and a giant-cell tumor, whereas Geschickter considers it as a giant-cell variant of bone cyst (Fig. 302).

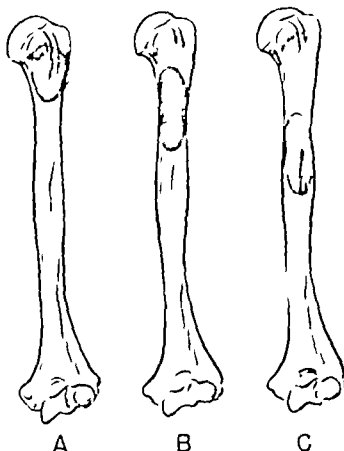


FIG. 302. Forms of bone cysts. (A) intermediate form between bone cyst and giant cell tumor found during adolescence. (B) bone cyst appearing in childhood. (C) latent bone cyst encountered in older children and adults.

ORIGIN

Since Virchow's first description of this lesion in 1878, numerous confusing and contradictory concepts of origin have been postulated; each one lacks incontestable evidence for acceptance.

Some investigators maintain that these lesions result from metabolic disturbance (Sisk), a theory not substantiated by other workers; yet other investigators have proposed that the disease results from intramedullary inflammation (Bloodgood, Pfeiffer)—however, bacteriologic investigation has failed to provide conclusive evidence to support this theory. Trauma and hemorrhage have aroused the interest of many workers who have attempted to correlate these factors with formation of bone cysts (Mauclaire and Burnier, Pommer, Jenckel and Mikulicz).



FIG. 303 Bone cyst in the upper end of the humerus of a female aged 13. Note the expansile nature of the lesion, the thin cortex and the juxta-epiphyseal location. No roentgenographic evidence of repair exists.

Geschickter and Copeland maintain that the contents of bone cysts (osteitis fibrosa) are closely related to giant-cell tumors and represent a reparative process of bony elements taking place around an area of bone destruction which soon becomes arrested although the healing or reparative phase may persist for many years giving rise to the latent bone cyst. Failure to collapse the bony walls of the cyst is responsible for persistence of the lesion into adult life. This fact is substantiated by the knowledge that fracture of the cortical shell, spontaneous or surgical usually is followed by rapid healing, complete obliteration of the cyst may occur.

AGE, INCIDENCE AND SITE

Bone cysts are encountered between the ages of 5 and 15 years, the intermediate variety (Coley) or giant-cell variant (Geschickter and Copeland) during adolescence while the latent inactive form is observed in older individuals in whom the lesion has a much longer history or has caused no symptoms.

Approximately 50 per cent of all cases of bone cysts are located in the upper ends of the humerus and the femur. Males are affected more frequently than females. The highest incidence occurs between 6 and 10 years, the age ranges from 3.5 to 60 years, the average being 16.4 years.

A characteristic feature of active bone cysts which distinguishes them from giant cell tumors is their metaphyseal location always on the diaphyseal side of the epiphyseal line. The quiescent or latent variety also originates in this area but gradually migrates toward the midshaft as the bone grows.

PATHOLOGY

Macroscopic study reveals that the contents of different bone cysts varies considerably. The duration of the lesion and the age of the patient are partially responsible for this variation. Acute bone cysts of short duration possess a thin enveloping bony shell. According to Bloodgood's classification a bone cyst may or may not possess a connective-tissue lining; the contents of the cyst may comprise a cavity filled with a yellowish fluid or a solid mesh of fibrous tissue (osteitis fibrosa). Occasionally numerous small cavities are encountered (multilocular cysts).

It is Coley's opinion that the neoplasm is initiated by a subcortical hemorrhage followed first by localized bone destruction, then granulation tissue containing osteoclasts (giant cells), fibrosis and finally cyst formation. Geschickter and Copeland describe the sequence of the process as first the formation of giant-cell areas followed by the appearance of new blood vessels and hemorrhage. Absorption of the hemorrhage,



FIG 304 (*Left*) Recent spontaneous fracture through cyst wall of a male aged 14. No clinical indications of the lesion existed prior to fracture (J. Edelken, Mt. Sinai Hospital, Philadelphia) (*Right*) Spontaneous fracture through cyst wall of a female aged 13. Observe reparative process initiated by the fracture. Note the periosteal bone formation bridging the fracture line (A. Price, Jefferson Medical College)

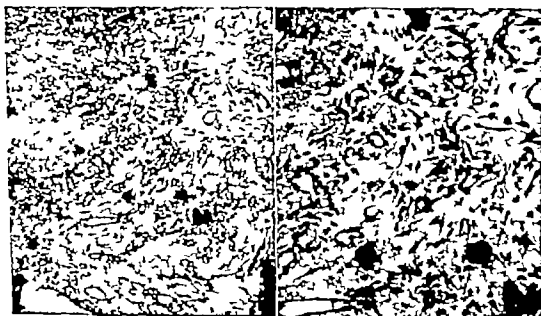


FIG 305 Contents of a bone cyst. Multinucleated giant cells are scattered throughout the tissue which simulates osteitis fibrosa. The tissue abounds in endothelial cells and fibroblasts (Photomicrographs $\times 125$ and $\times 250$)

in turn results in the formation of cysts lined with connective tissue which ultimately is transformed into bone (Fig 303)

Generally progression of bone cysts is slow its rate of growth is in accordance with the severity of the symptoms. The affected bone portion slowly expands as the overlying cortex becomes very thin. No evidence of bone reaction is discernible in the more acute types although some condensation of the bony shell is demonstrable in the latent forms. Fracture may ensue following minor trauma to the weakened bone. Fracture stimulates healing as evidenced by rapid new bone formation. In some instances, the healing following fracture may obliterate the cyst.

Microscopic study discloses wide variation in the histologic structure of bone cysts. Some cases bear a close resemblance to giant-cell tumors; this is particularly true of the intermediate varieties observed during adolescence. Other cysts do not differ from osteitis fibrosa. It is apparent that the variations mentioned are different stages of the same pathologic process and are related to giant-cell tumors. Many investigators maintain that the variable factors responsible for the different behaviors of bone cysts and giant-cell tumors include the age of the patient, the region of the bone affected and the response of the surrounding bone to the pathologic process. In the metaphysis sufficient resistance is encountered to limit the aggressiveness of the lesion while in the epiphyseal region less resistance is offered by the cancellous bone thereby allowing the destructive phase to progress. Hence, in the metaphysis the process results in bone cysts while giant-cell tumors result in the epiphyseal region.

CLINICAL FEATURES

Not infrequently bone cysts are discovered accidentally through roentgenograms taken for reasons not related to the affected region. The first indication of a bone cyst in children may be a pathologic fracture following minor trauma. In general any

symptoms present are mild, varying in duration from a few months to many years. Bone cysts found in adolescence run a more acute clinical course, the duration being under 6 months. Latent cyst may date back from 20 to 40 years, with an average duration period of 2½ years.

Pain. Pain is seldom a prominent clinical feature. It may occur only after exertion and may be relieved by rest. Of course, the symptom is accentuated when fracture occurs. Pain is usually in the region of the upper end of the humerus, occasionally, it is referred posteriorly to the scapular region. Firm pressure over the affected bone invariably elicits moderate tenderness.

Swelling. This sign is rarely detectable because of the fleshy deltoid muscle covering the upper end of the humerus. Palpation of the area, particularly in more progressive lesions, reveals a fusiform enlargement of the upper end of the humerus as compared with the opposite side.

Impaired Function. Function of the shoulder joint is seldom impaired. However, it is obvious that disability is increased when fractures occur.

ROENTGENOLOGIC FEATURES

The distinctive features of bone cysts are (1) a destructive lesion in the metaphysis in growing individuals (before closure of the epiphyseal line), (2) uniform expansion of the metaphyseal region of the shaft, (3) a smooth thin cortical shell and (4) the occasional occurrence of irregular trabeculae throughout the bone defect.

These features may be absent in acute early lesions. The lesion on the radiographs may appear as a radiolucent area in the metaphysis close to the epiphyseal disk. Lack of bone reaction is a characteristic finding in all uncomplicated bone cysts. Radiographic evidence of new bone formation is readily discernible in case of fractures. Also as previously noted some bone condensation is demonstrable around a latent cyst.

DIAGNOSIS

The age of the individual, the location of the lesion in the metaphysis (on the distal physical side of the epiphyseal plate) roentgenologic characteristics and a mild clinical course are sufficient to establish a diagnosis of bone cyst. Lesions which occasionally require consideration in diagnosing this neoplasm are giant-cell tumor, benign chondroblastoma and chondroma.

Giant-cell tumors occur in older individuals (after 20 years of age) when the upper epiphysis has united to the shaft of the humerus. They are located in the epiphysis, starting from an eccentric position and extending centrally and toward the articular cartilage.

Benign chondroblastomas usually are confined to the region of the greater tuberosity; they do not cross the proximal or distal epiphyseal plates; they have a fluffy, mottled appearance; and histologic studies reveal that they consist essentially of immature cartilage.

Chondromas may bear a close resemblance to bone cysts; however, they are rarely found in long bones.

TREATMENT

There is general agreement that surgical intervention is the treatment of choice. Implantation of bone chips or longer segments of bone parallel with the long axis of the cyst ensures more rapid regeneration of bone and minimizes the possibilities of spontaneous fracture through the affected area. Although pathologic fractures initiate healing which occasionally obliterates the bone cyst, curettage of the cavity and reinforcement with bone chips assures more prompt healing. The surgical technique employed in the treatment of these lesions is similar to that of giant-cell tumors (see pp 333).

Irradiation of bone cysts is not warranted because it may damage the epiphysis or even result in premature ossification of the epiphyseal disk, thereby interfering with normal bone growth which in turn may

result in the shortening of the humerus. Also the possible development of sarcoma in irradiated bone is a definite contraindication to the use of roentgen rays in cases where other forms of therapy are more effective.

Recurrences of the bone cyst subsequent to curettage is not a frequent occurrence. A second curettage or in rare instances even a third may be necessary to achieve a cure.

SARCOMAS OF THE BONE

INCIDENCE AND AGE

In 1922, Codman made a survey of bone sarcoma in Massachusetts and estimated an incidence of one case in every 100,000 inhabitants at any one time. According to Coley, there is an incidence of one case of osteogenic sarcoma for every 117,000 of the population in the United States. Sarcoma of bone is a little more prevalent in males than in females.

As previously stated, half of all cases occur in the second decade and two-thirds occur between the ages of 10 and 30 years. In patients over 50 years of age, osteogenic sarcoma usually indicates the existence of Paget's disease of bone with sarcomatous degeneration.

SITE

Over a tenth of all bone sarcomas are found in the bones of the shoulder girdle (Codman records 13.5 per cent), the humerus being the most frequent location. The clavicle is rarely affected. As in all tubular bones, the preferred site in the humerus is in the metaphyseal region.

GRADATION OF DEGREE OF MALIGNANCY

Some investigators believe that it is possible to determine the degree of malignancy of sarcomas of bone by the histologic characteristics of the cells and the tissues comprising the neoplasm. Broder has evolved a classification of gradation based on the differentiation of cells. Some workers hesitate to place too much faith on such a gradation while others rely heavily on the

pathologist's classification before instituting definitive therapy for a specific lesion.

Meyerding is convinced that the grading of malignant tumors has reached a high degree of proficiency at the Mayo Clinic. The decision of the pathologist therefore, plays a major role in the choice of treatment or the extent of surgery performed. He states

When after thorough study of the sections of tissue the pathologist reports a Grade I osteogenic sarcoma, I feel that there may be some merit in performing a more conservative type of operation whereas when he reports a Grade 4 or higher degree malignant lesion I would without hesitation perform amputation of the involved extremity. In some cases it is possible to excise in toto the portion of bone containing the tumor and insert a bone graft without the danger of local recurrences or metastasis.

The value of grading as an aid in prognosticating the course of neoplasms is apparent. Coley notes that the percentage of 5 year survivals in low grade tumors is more than twice that of the higher grades combined. However he does not believe that fine subdivisions of grading into numerous groups is of any practical significance.

OSTEOGENIC SARCOMA

Essentially this group comprises tumors originating in bone and capable of producing bone. It makes up over 50 per cent of all bone sarcomas. The character and the amount of bone produced varies in different tumors giving rise to several clinical types. Generally the outlook is unfavorable and the mortality is high. In the humerus as in other long bones the tumors originate in the metaphysis and tend to grow outward under the deltoid and downward into the shaft of the humerus. As a rule they do not invade the head of the humerus or break through into the capulohumeral joint. Invasion of the medullary canal and destruction of the cancellous and cortical bone is a characteristic feature of these neoplasms.

Stimulation of the periosteum by the advancing tumor may lead to varying amounts of new bone formation in the shaft distal to

the tumor (Codman's triangle). With dissolution and perforation of the cortex the tumor mass may progress beyond the limits of the bone varying degrees of ossification may occur in the tumor outside the shaft. Early metastasis to the lungs is the rule, generally, dissemination of the tumor is achieved via the blood stream, but occasionally by the lymphatic.

Pathologic fracture of the humerus is not infrequent. Of 24 cases of osteogenic sarcoma of the upper end of the humerus reviewed by Codman (1930), 19 exhibited fractures through the involved area of the humerus. Coley and Sharp (1930) observed that pathologic fracture was more common in the humerus than in the femur despite the fact that the latter is a weight bearing bone. (Four of the five cases which had not sustained a fracture were classified as sclerosing osteogenic sarcomas.)

SCLEROSING OSTEOGENIC SARCOMA

This form is observed most commonly in the lower extremity. Although it is encountered not infrequently in the upper end of the humerus it is rarely observed in the scapula and the clavicle. Ages ranged from 8 to 61 years the average being 19.3 years. Males were affected more frequently than females. The highest incidence is found between 11 and 15 years.

In the humerus as in other long bones the tumor arises in the metaphyseal region. Its clinical course is not so acute as the osteolytic form the duration of symptoms from onset to treatment being less than a year. (In contrast with other varieties of osteogenic sarcoma, pathologic fracture is rare.)

Palpation in advanced cases reveals an irregular hard bony tumor in the upper end of the humerus. The deltoid may partly conceal the bony mass invariably however the deltoid region appears fuller and more prominent than the unaffected side. The cases observed by the author exhibited some impairment of shoulder function. As a

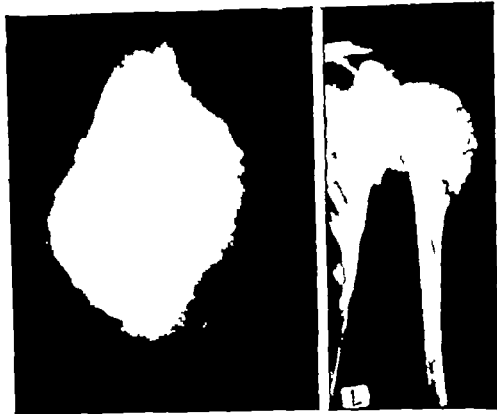


FIG 306 Sclerosing osteogenic sarcoma of humerus (*left*) Patient developed this primary lesion after a similar primary lesion had developed in the femur (*right*) which was treated by amputation (Coley Neoplasms of Bone, New York Hoeber p 239)

rule motion was guarded, and movements of the arm were kept below the horizontal even though the disability was not severe

The roentgenographic features of this tumor are characteristic. Irregular areas of increased density obliterating the pattern of normal bone appear in the metaphyseal region of the humerus. As the tumor grows the cortex is destroyed and the soft tissues are invaded the same dense areas of tumor bone appearing beyond the confines of the shaft of the humerus. The tumor rarely crosses the epiphyseal line to involve the humeral head. Fine spicules of tumor bone radiating at right angles to the shaft of the humerus may be seen giving the 'sun ray' appearance, believed by many to be a pathognomonic feature of malignant bone sarcoma. However this peculiar 'sun ray' occurs in other affections of bone (Fig 306)

TELANGIECTATIC SARCOMA OF THE BONE (OSTEOLYTIC TYPE OF OSTEOGENIC SARCOMA OR MALIGNANT BONE ANEURYSM)

This tumor is relatively rare and its origin is not definitely established. Diagnosis may be exceedingly difficult, this tumor



FIG 307 Osteolytic form of osteogenic sarcoma involving the entire upper end of the humerus. Note complete dissolution of bony elements and extension of tumor tissue into the surrounding soft tissues. (Geschickter and Copeland Tumors of Bone ed. 3 Philadelphia Lippincott, p 217)



FIG 305. Rare type of osteogenic sarcoma (medullary fibrosarcoma) of the upper end of the humerus. Note the irregular destruction of the cancellous bone extending distally in the medullary canal; the head of the humerus is spared. In this instance there is no radiographic evidence of periosteal bone formation.

often being confused with bone cysts, giant cell tumors and metastatic carcinoma. It is a highly destructive vascular lesion, very malignant, and carries an unfavorable outlook. Telangiectatic bone sarcomas are encountered more frequently in the second decade of life but may occur in older age groups. Pathologic fracture is a common sequela. Some series report an incidence of approximately 50 per cent. Ages range from 13 to 60 years, the average being 28.5 years. The highest incidence occurs between 16 and 20 years.

The shoulder region exhibits a fusiform or globular swelling of the proximal end of the arm; the skin may be stretched tightly over the tumor but it may remain freely

movable; the surface temperature of the affected area may be increased. The tumor may be soft and boggy. Shoulder function is usually impaired somewhat.

Roentgenologic studies reveal pronounced destruction as if the bony elements of the upper end of the humerus were melting away. The articular head is usually spared, and the tumor does not invade the joint cavity. However, in advanced cases the head of the humerus and the joint cavity may be implicated. Early cases may show new periosteal bone formation on the shaft distal to the tumor and even sun ray spicules. Later, with tumor progression, no evidence of new bone formation may be discernible. Pathologic fracture, as noted above, is a frequent sequela (Fig. 307).

MEDULLARY AND SUBPERIOSTEAL OSTEOGENIC SARCOMA

This subdivision comprises a relatively common group of osteogenic sarcomas which originate within the bone, destroy first the spongiosa and then the cortex. Tumor tissue grows and extends into the medullary canal and occupies a position under the raised periosteum.

Tumor bone forms varying in amount and degrees of differentiation. Reactive new bone formation is laid down by the periosteum distal to the tumor in an attempt to obstruct its progression. As the tumor grows, reactive bone is destroyed and nature lays down another layer distal to the limits of the tumor. Again, as in other types, the humeral head is not invaded; the tumor does not extend beyond the epiphyseal line.

FIBROSARCOMA OF BONE

It is now generally conceded that fibrosarcoma may arise from the fibrous connective tissue or from the tissue forming the outer layers of the periosteum. The neoplasms never form bone; they consist chiefly of spindle cells whose degree of malignancy depends upon the degree of cell differentiation. In general, they are considered the



FIG 309 (*Top left*) Fibrosarcoma (periosteal sarcoma) in right shoulder of a male adolescent. Note that the shaft of the humerus appears not to be implicated, also the lack of any new bone formation. (B. P. Widmann and J. C. Howell, Philadelphia General Hospital.) (*Top right*) Later stage of same tumor; the shaft of the humerus is still intact showing only minimal alterations. (Widmann and Howell.) (*Bottom left*) Final stage exhibits complete destruction of the upper end of the humerus. Observe the absence of new bone in the tumor mass. Other similar lesions developed in opposite shoulder (*bottom right*) and in both femurs. This was believed to be an instance of multiple primary sarcomas. (Widmann and Howell.) (*Bottom right*) Left shoulder of patient exhibited in the other figures. Note the extensive tumor without apparent involvement of the shaft of the humerus.



least malignant of the osteogenic sarcomas and offer the most favorable prognosis

Medullary fibrosarcomas originate in the cancellous bone near the epiphyseal line. The tumor invades the spongiosa eroding

EWING'S SARCOMA

INTRODUCTION

Ewing's tumor is now accepted as a definite entity with characteristic clinical,

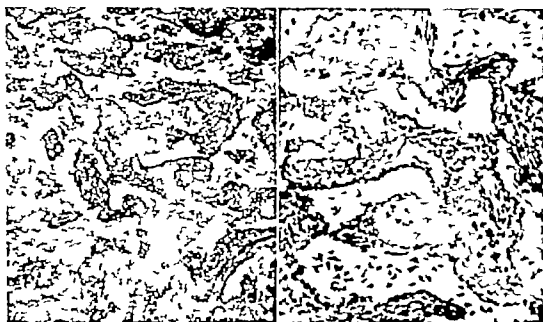


FIG 310 Osteogenic sarcoma (osteoblastic form) ($\times 125$ and $\times 250$) Observe the formation of irregular osteoid trabeculae whose peripheries are lined by numerous malignant osteoblasts. Also note that the osteoid tissue lacks a definite pattern—it is laid down in a disorderly fashion; numerous vascular channels are dispersed throughout the tissue.

and perforating the cortex. The elevated periosteum may lay down reactive bone around and distal to the tumor. Radiographically there is no evidence of tumor bone formation; hence the central area of destruction in the end of the shaft is depicted as an irregular radiolucent area sometimes confused with the more malignant osteolytic sarcoma or with a central chondroma (Fig 308).

Fibrosarcoma originating from the outer layers of the periosteum does not form bone and it exhibits little tendency to invade the adjacent bone. However, sometimes the bone is involved, thus making it difficult or impossible to determine the origin of the neoplasm (Fig 309). Fibrosarcomas are amenable to surgery and it is agreed that if they were treated early, the survival rate of this group would be increased greatly. Unfortunately, in most instances, treatment is initiated only after metastasis

radiographic and histologic features. However, there is much controversy as to its histogenesis. Ewing described the neoplasm as consisting of angio-endothelial cells and believed it to be of endothelial origin. Jaffe and Lichtenstein are of the opinion that it is derived from primitive marrow-connective tissue. Stout believes that the tumor is a variant of reticulum sarcoma originating from the supporting mesenchymal tissue of the bone marrow, while Oberling states that the tumor is a reticulosarcoma.

In spite of its obscure and undetermined origin, many distinctive features of this bone neoplasm have been uncovered. It is exceedingly radiosensitive, metastasizes to lung, lymph nodes, and other bones, and usually terminates fatally.

INCIDENCE, AGE, SEX, AND SITE

This primary malignant bone tumor is relatively common and occurs most fre-

quently in children and young adults. It is rarely encountered after 30 years of age. The bones of the shoulder girdle are frequently involved and although it is usually observed in the shafts of long bones, it not

CLINICAL FEATURES

Pain with or without trauma is usually the first manifestation of the disease, and its onset is insidious in most instances. It



FIG. 311 Ewing sarcoma involving upper one-half of the humerus in a male 11 years of age. Observe the reactive periosteal new bone formation giving the shaft a laminated appearance and the increased density within the shaft indicative of new endosteal bone formation. Radiating spicules of new bone are also discernible. The epiphysis is not implicated. (Geschickter and Copeland: *Tumors of Bone*, ed. 3, Philadelphia: Lippincott 1949, p. 416.)

Infrequently arises in the upper end of the humerus. Males are affected more frequently than females, the ratio being approximately 2 to 1. The age ranges from 4 to 44 years, the highest incidence being between 6 and 15 years.

is not severe at first and it may be intermittent in nature. Later the pain is constant, being most severe at night. Swelling is always found, being readily demonstrable in the bones of the shoulder joint. Marked impairment of function seldom occurs, even

least malignant of the osteogenic sarcomas and offer the most favorable prognosis.

Medullary fibrosarcomas originate in the cancellous bone near the epiphyseal line. The tumor invades the spongiosa eroding

EWING'S SARCOMA

INTRODUCTION

Ewing's tumor is now accepted as a definite entity, with characteristic clinical

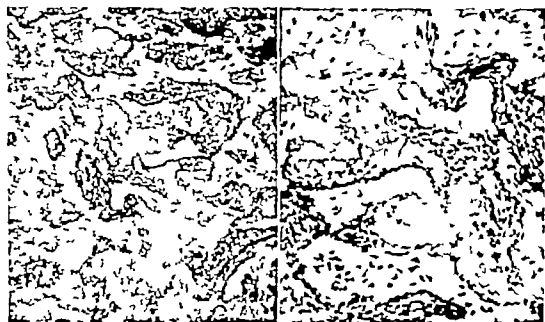


FIG 310 Osteogenic sarcoma (osteoblastic form) ($\times 125$ and $\times 250$) Observe the formation of irregular osteoid trabeculae whose peripheries are lined by numerous malignant osteoblasts. Also note that the osteoid tissue lacks a definite pattern. It is laid down in a disorderly fashion. Numerous vascular channels are dispersed throughout the tissue.

and perforating the cortex. The elevated periosteum may lay down reactive bone around and distal to the tumor. Radiographically there is no evidence of tumor bone formation; hence the central area of destruction in the end of the shaft is depicted as an irregular radiolucent area, sometimes confused with the more malignant osteolytic sarcoma or with a central chondroma (Fig 308).

Fibrosarcoma originating from the outer layers of the periosteum does not form bone and it exhibits little tendency to invade the adjacent bone. However, sometimes the bone is involved, thus making it difficult or impossible to determine the origin of the neoplasm (Fig 309). Fibrosarcomas are amenable to surgery, and it is agreed that if they were treated early, the survival rate of this group would be increased greatly. Unfortunately, in most instances, treatment is initiated only after metastasis.

radiographic and histologic features However, there is much controversy as to its histogenesis. Ewing described the neoplasm as consisting of angio-endothelial cells and believed it to be of endothelial origin. Jaffe and Lichtenstein are of the opinion that it is derived from primitive marrow-connective tissue. Stout believes that the tumor is a variant of reticulum sarcoma, originating from the supporting mesenchymal tissue of the bone marrow, while Oberling states that the tumor is a reticulosarcoma.

In spite of its obscure and undetermined origin, many distinctive features of this bone neoplasm have been uncovered. It is exceedingly radiosensitive, metastasizes to lung, lymph nodes, and other bones, and usually terminates fatally.

INCIDENCE AGE SEX AND SITE

This primary malignant bone tumor is relatively common and occurs most fre-

quently in children and young adults. It is rarely encountered after 30 years of age. The bones of the shoulder girdle are frequently involved and although it is usually observed in the shafts of long bones it not

CLINICAL FEATURES

Pain with or without trauma is usually the first manifestation of the disease and its onset is insidious in most instances. It



FIG. 311 Ewing sarcoma involving upper one-half of the humerus in a male 11 years of age. Observe the reactive periosteal new bone formation giving the shaft a laminated appearance and the increased density within the shaft indicative of new endosteal bone formation. Radiating spicules of new bone are also discernible. The epiphysis is not implicated (Geschickter and Copeland, *Tumors of Bone*, ed. 3, Philadelphia: Lippincott 1949, p. 416.)

infrequently arises in the upper end of the humerus. Males are affected more frequently than females, the ratio being approximately 2 to 1. The age ranges from 4 to 44 years, the highest incidence being between 6 and 15 years.

is not severe at first and it may be intermittent in nature. Later the pain is constant, being most severe at night. Swelling is always found, being readily demonstrable in the bones of the shoulder joint. Marked impairment of function seldom occurs even

least malignant of the osteogenic sarcomas and offer the most favorable prognosis

Medullary fibrosarcomas originate in the cancellous bone near the epiphyseal line. The tumor invades the spongiosa, eroding

EWING'S SARCOMA

INTRODUCTION

Ewing's tumor is now accepted as a definite entity, with characteristic clinical,

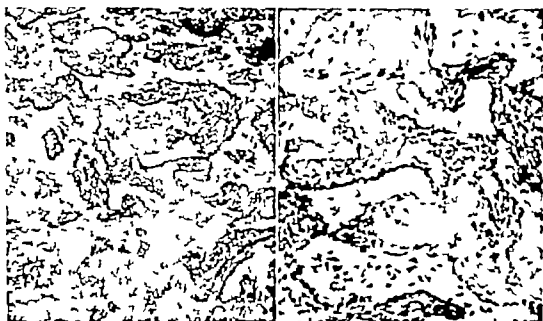


FIG. 310 Osteogenic sarcoma (osteoblastic form) ($\times 125$ and $\times 250$) Observe the formation of irregular osteoid trabeculae whose peripheries are lined by numerous malignant osteoblasts. Also note that the osteoid tissue lacks a definite pattern—it is laid down in a disorderly fashion; numerous vascular channels are dispersed throughout the tissue.

and perforating the cortex. The elevated periosteum may lay down reactive bone around and distal to the tumor. Radiographically, there is no evidence of tumor bone formation; hence the central area of destruction in the end of the shaft is depicted as an irregular radiolucent area, sometimes confused with the more malignant osteolytic sarcoma or with a central chondroma (Fig. 308).

Fibrosarcoma originating from the outer layers of the periosteum does not form bone and it exhibits little tendency to invade the adjacent bone. However, sometimes the bone is involved, thus making it difficult or impossible to determine the origin of the neoplasm (Fig. 309). Fibrosarcomas are amenable to surgery, and it is agreed that if they were treated early, the survival rate of this group would be increased greatly. Unfortunately, in most instances, treatment is initiated only after metastasis.

radiographic and histologic features However, there is much controversy as to its histogenesis. Ewing described the neoplasm as consisting of angio-endothelial cells and believed it to be of endothelial origin. Jaffe and Lichtenstein are of the opinion that it is derived from primitive marrow-connective tissue. Stout believes that the tumor is a variant of reticulum sarcoma, originating from the supporting mesenchymal tissue of the bone marrow, while Oberling states that the tumor is a reticulosarcoma.

In spite of its obscure and undetermined origin, many distinctive features of this bone neoplasm have been uncovered. It is exceedingly radiosensitive, metastasizes to lung, lymph nodes, and other bones, and usually terminates fatally.

INCIDENCE AGE SEX AND SITE

This primary malignant bone tumor is relatively common and occurs most fre-

they are very poor. Resection holds no more hope for the patient but he is spared a mutilating operation. Moderate doses of roentgen therapy before amputation or resection and heavier dosages after the wound has

Lack of such systemic reactions as leukocytosis and consistent negative bacteriologic and histologic studies tend to discredit the infectious theory. Trauma plays no part in the histogenesis of the tumor. It is often

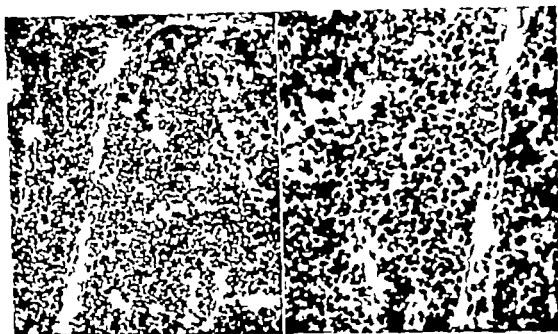


FIG. 312 Ewing's sarcoma. Observe the compact areas of small polyhedral cells with a hyperchromatic nucleus and scanty cytoplasm. No intercellular stroma is present. (Photomicrographs $\times 125$ and $\times 250$)

healed is the plan of treatment in most institutions.

Prognosis on the whole is poor; metastases and death are the usual sequelae. The survival period after the initial symptoms of the disease appear varies but it is rarely more than two years.

OSTEOID OSTEOMA

INTRODUCTION

Osteoid osteoma is now generally accepted as a distinct clinical entity. It has been encountered in all the bones of the body except the cranial bones, the scapula and the clavicle. Most investigators agree with Jaffe that it is a benign neoplasm of bone. However, a few refuse to accept this concept, believing that the lesion is a chronic bone infection or abscess (Brown and Ghormley, Brailsford). The weight of evidence is against the infectious theory.

confused with Brodie's abscess and Garre's sclerosing osteitis.

AGE, SEX AND INCIDENCE

The neoplasm is more prevalent in males than in females. It has a predilection for adolescents and young adults, although Sherman reports one instance in an individual 51 years old. The highest incidence is found between the ages of 10 and 20 years.

SITE

Sherman reported 158 cases of osteoid osteoma, 128 of which were gathered from the literature. The lesion was found in all bones of the skeletal system except the cranial bones, the scapula and the clavicle. Ten of the cases (6.2 per cent) were encountered in the humerus. The most frequent site is the tibia and the femur. It may be observed in the cancellous bone near or

with extensive involvement. Pathologic fracture is more frequent in the upper end of the humerus than elsewhere

Constitutional reactions such as fever and leukocytosis are common manifestations of the disease responsible, in many cases, for the erroneous diagnosis of osteomyelitis. Not infrequently such cases have been operated upon and the diagnosis has not been established until after meddlesome surgery had been performed

The disease tends to pursue a variable clinical course and is usually fatal. Metastases may take place early in the lungs and the lymph nodes, later in other bones and viscera. The bones of the skull are frequent sites for metastatic lesions. Reported cases reveal that the time range between the first appearance of bone lesion and multiple bone involvement ranges from 2.5 months to 4 years

Ewing's sarcoma is osteolytic in nature and never produces bone. The new bone formation noted radiographically is reactive bone formed by the periosteum and endosteal elements through stimulation by the advancing tumor. In the early stages increased density and expansion of the shaft of humerus may be discernible. This results from endosteal and subperiosteal bone formation. Subperiosteal bone is laid down parallel with the shaft giving the onion peel effect often described as characteristic of Ewing's tumor

Later the tumor extends along the shaft of the humerus with destruction of the cortex irregular mottling and osteoporosis of the medullary cavity and increased density of the tumor's periphery resulting from periosteal bone formation. Reactive bone formation is usually more abundant in children than in adults. In advanced lesions dense spicules of necrotic bone may be demonstrable simulating an infectious process of bone (Fig. 311)

PATHOLOGY

The tumor arises in the medullary canal. It destroys and infiltrates cancellous bone

and then cortical bone. It extends up and down the medullary canal the shaft expanding as it grows. With destruction of the cortex, the tumor proliferates beneath the periosteum. Large amounts of tumor tissue are found in this position in advanced cases. Marked thickening of the shaft may result from endosteal and subperiosteal bone formation. Only rarely does the tumor cross the epiphyseal line. Subperiosteal bone is laid down parallel with the shaft but, with separation of the periosteum, new bone is formed at right angles to the shaft following the course of the blood vessels (Ribbert)

Histologic study reveals that the tumor consists of compact areas of small polyhedral cells with a round hyperchromatic nucleus and scanty cytoplasm with distinct but slightly irregular outline. No intercellular stroma is present. Microscopic interpretation of this lesion is most often confused with reticulum-cell sarcoma and metastatic bone lesions from neuroblastoma of the sympathetic system. Willis is of the opinion that frequently the latter is diagnosed erroneously as Ewing's tumor (Fig. 312)

PROGNOSIS AND TREATMENT

Ewing's tumors are very radiosensitive, but irradiation alone never has effected a cure. Roentgen ray therapy relieves pain and causes regression of the primary tumor. However it does not prevent metastasis, which invariably follows regardless of the local status of the initial neoplasm. Inadequate dosages of irradiation tend to make the tumor less radiosensitive

Surgical intervention with or without irradiation is accepted as the best form of therapy. Coley states that resections should be performed more frequently in such bones as the fibula, the ulna and even the scapula and the humerus. He justifies this opinion by the fact that amputation offers no more hope of cure than does resection.

Statistics reveal that the results of amputation are far from encouraging. In fact

of the entire circumference of the shaft. Early cases may fail to reveal any radiographic findings, however, subsequent examinations will uncover the true nature of the disease.

PATHOLOGY

Gross study of the nidus reveals it to be a small cavity usually under 2 centimeters in diameter and containing friable, reddish brown substance. Microscopic examination discloses a highly vascular connective tissue stroma in which are found trabeculae of osteoid tissue some of which is irregularly calcified to form osseous tissue. Multi nucleated giant cells may be present (Fig 313).

TREATMENT

The most effective form of therapy now known is block resection of the nidus and a portion of the surrounding bone. If the lesion is completely removed pain disappears immediately and recurrence of the symptoms does not occur. This is not true if the nidus and its contents are not totally removed as occasionally occurs following curettage or inadequate block resection. The train of symptoms is not interrupted with incomplete removal. Even upon awakening from the anesthesia the patient will continue to complain of the same type of pain. If the defect is large in tubular bones (such as the humerus) postoperative immobilization of the extremity should be insisted upon to prevent fracture through the operative site until a sufficient amount of new bone is formed. This period may be shortened by filling in the bone defect with bone chips.

The author has seen such an unfortunate complication following block resection of an osteoid osteoma in the radius. Sufficient data have not been accumulated to predict the outcome of untreated cases. Jaffe is of the opinion that spontaneous clinical arrest occurs after many years. Sherman records one case under Phemister's observation in which the diagnosis of osteoid osteoma by

the present-day criterion was apparent. The patient refused surgery, pain and tenderness subsided gradually and 7 years later had disappeared completely. Radiographic examination 24 years later revealed persistent thickening and sclerosis of the cortex; the nidus was not visible. The patient was completely free of all symptoms.

CHONDROSARCOMA

INTRODUCTION

Chondrosarcoma was included in the general classification of osteogenic sarcoma up to 1939. However, a realization that these tumors arise from cartilage, the greater portion of or even the entire tumor consisting of cartilage and exhibiting varying degrees of differentiation, plus the knowledge that they pursue a more protracted clinical course than most osteogenic sarcomas, forced the Committee of the Registry of Bone Sarcoma to remove these neoplasms from the general classification of osteogenic sarcomas and assign them to the separate division "chondrosarcoma." They were further subdivided into (1) primary chondroblastic sarcoma and (2) secondary chondrosarcoma.

PRIMARY CHONDROBLASTIC SARCOMA

These tumors have a predilection for the ends of the long bones and are encountered most frequently (in order of frequency) in the region of the knee, the shoulder and the hip. They arise in the cancellous bone from cartilage in the region of the epiphyseal plate. Primary chondroblastic sarcomas are exceedingly malignant and do not differ essentially in clinical course and prognosis from osteolytic osteogenic sarcoma.

Clinical Features. Like other osteogenic sarcomas, primary chondroblastic sarcomas usually occur in the second decade of life, the highest incidence being between 10 and 15 years. The shoulder region is a frequent site of all primary chondroblastic sarcomas.

Pain, swelling and impaired function are the cardinal clinical features. Pain may be mild and is often associated with some form

at a distance from the articular cartilage, or it may occupy an intracortical or a subperiosteal position

CLINICAL FEATURES

Pain is the cardinal clinical manifestation of this disease. The author has observed one case of osteoid osteoma in the middle

lar surface of the humeral head. Reported cases disclose that pain may be present from 2 months to 2 or more years before relief is sought.

In those areas in which there is little overlying soft tissue, swelling and thickening of the bone may be palpable. Point tenderness over the affected bony area is a



FIG. 313. Osteoid osteoma. The tissue is rich in fibroblasts and osteoblasts forming osteoid spicules. (Photomicrographs $\times 32$ and $\times 16$.)

third of the humerus. At first the pain which had existed 8 months before the patient sought medical advice was mild and vague, localized in the region of the deltoid tubercle. It was relieved by rest and accentuated by activity. Later the pain became more constant and was noticed to be centered on the anterolateral aspect of the humerus. Nocturnal pain is a common complaint. In the instance just mentioned the patient's sleep was often interrupted by excruciating pain. Sherman reports several cases in which impaired function occurred in the adjacent joint, fluid being also demonstrable within the joint, simulating a primary arthritis.

The author has observed these findings in two cases involving the distal end of the radius in which the wrist joint was affected and one case involving the proximal phalanx of the index finger. In the last instance there was pronounced swelling of the metacarpophalangeal joint. However, these findings were not noted in the lesion involving the humerus, probably because the diseased area was at some distance from the articu-

lar surface of the humeral head. Such local evidence of inflammation as redness, increased local temperature and edema of the soft tissues is absent—as are other systemic manifestations of infection, such as fever and leukocytosis.

ROENTGENOLOGIC FEATURES

Diagnosis is usually made through the distinctive radiographic characteristics. In most instances a small round or ovoid radiolucent area surrounded by dense sclerotic bone is discernible. The central rarefied area or nidus is sometimes demonstrable only in laminographic examination. However, the dense sclerotic bone is always visible. The extent of this sclerotic hypertrophied bone varies with the location and the duration of the lesion. Lesions of long duration in the cortex or near the periosteum are usually associated with pronounced regional thickening of the shaft, while those located in the spongiosa are surrounded by a circumscribed area of bone of varying density which is seldom marked. Occasionally intracortical lesions cause thickening

throughout which may be found cystic and hemorrhagic areas. Its consistency varies according to the type of cartilage and the amount of calcification and ossification plus the extent of tissue degeneration.

Microscopic examination reveals pronounced variation in different tumors, also in different areas of the same tumor. The bulk of the tumor comprises cartilage cells

exhibiting varying stages of differentiation and calcification a cartilaginous matrix and varying amounts of myxomatous tissue. The stage of differentiation of the cartilage tissue indicates the degree of malignancy. Neoplasms exhibiting the least differentiation are the most malignant (Figs. 315 and 316).

Treatment and Prognosis. Surgery still

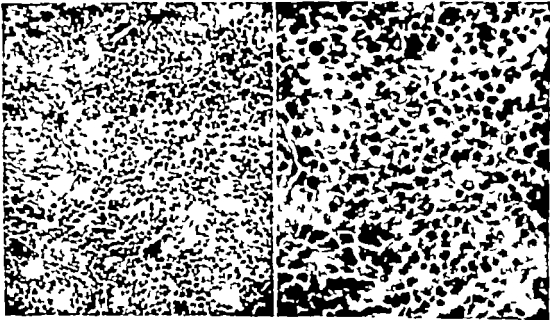


FIG. 315 Chondroblastic sarcoma. Note the pleomorphic nature of the chondroblasts, marked vascularity of the tissue and lack of stroma. (Photomicrographs $\times 125$ and $\times 250$.)



FIG. 316 Chondrosarcoma. Observe cellular pleomorphism, numerous double nucleated cells and loss of polarity of the chondroblasts. Although not numerous, several cells show mitotic figures. (Photomicrographs $\times 125$ and $\times 250$.)

of trauma early in the disease. The duration of symptoms before examination varies from 7 to 16 months. Pain becomes more constant and function at the shoulder becomes definitely impaired as the lesion progresses. Spontaneous fractures rarely occur. Like other osteogenic sarcomas, rapidly growing tumors may show such severe bodily reactions as elevated temperature, loss of weight and leukocytosis. The lesion may assume extraordinary dimensions in the shoulder region. The swelling may have a doughy, elastic consistency at first; later it may become strong and hard with secondary calcification within the tumor.

Metastases to lungs via the blood stream occurs early in the disease. No proved cases of metastases by way of the lymphatic system have as yet been recorded.

Chondrosarcomas are radioresistant lesions; surgery has proved to be the most effective form of therapy. As pointed out by Coley, the serum phosphatase level in these lesions invariably is elevated and its range

is indicative of the activity of the tumor. If there are no metastases, the level diminishes following therapy.

Roentgenographic Features. Lesions beginning centrally are often depicted as irregular radiolucent areas, resulting from the destruction of the spongiosa and with no evidence of new bone formation or bone condensation around them. They involve both sides of the epiphyseal line and extend to the articular surface of the humeral head. Such lesions mimic giant-cell tumors, osteolytic osteogenic sarcomas and bone cysts. With destruction of the cortex and involvement of the periosteum, an area of dense bone may be seen around the lesion. Also elevation of the periosteum by the tumor may produce bone formation along the shaft distal to the lesion (Codman's triangle). The tumor may be traversed by coarse trabeculae giving it a multilocular appearance.

Pathology. Macroscopically the tumor consists of a grayish white material



FIG. 314 (*Left*) Unusual site for giant cell tumor (male 15 years old) resembling primary chondroblastic sarcoma of upper end of humerus (T. A. Shallow, Jefferson Medical College.) (*Right*) Material obtained from tumor shown at left. (Photomicrograph $\times 157$)

Roentgenographic Features Roentgenograms may disclose the remains of a chondroma or osteochondroma from which arises an irregular granular blotchy mass. Irregular areas of increased density in the

tumor are produced by islands of calcification and ossification. The tumor may cause extensive destruction of the cortex and invade the medullary canal; it may extend peripherally invading the soft tissue. Irreg-



FIG 317 (Top left) Female 53 years old, showing a chondrosarcoma which implicates the left scapula and the upper end of the humerus. Biopsy of the tumor was done elsewhere, 6 months after the tumor mass was first noted and the diagnosis of chondrosarcoma was made. The patient was treated erroneously by irradiation. (Top right) Roentgenograph of the lesion a year after treatment by irradiation. (Bottom left) Patient 7 days after interthoracoscopic amputation. (Bottom right) Photomicrograph ($\times 150$) of tissue obtained from the amputated extremity. Observe the pleomorphic character of the chondroblasts and the paucity of stroma.

offers more than any other form of therapy, provided that it is performed early. As a rule metastases has already taken place when surgery is instituted. Early amputation is the indicated procedure in all cases.

The outlook is generally grave. Resection of a chondroblastic tumor is rarely justified. However, it may be done in secondary chondrosarcomas which are shown by histologic studies to be of low grade malignancy and are located in bones which are amenable to resection. The prognosis in chondroblastic sarcomas about the shoulder appears to be more favorable than elsewhere in the body.

SECONDARY CHONDROSARCOMA

Malignant transformation of pre-existing benign cartilaginous lesions such as chondromas and osteochondromas (single or multiple) is not infrequent in bones of the shoulder girdle (Fig. 317). This is especially true of benign tumors which involve the scapula and the humerus. Secondary chondrosarcoma in patients over 50 years of age may arise in bone affected with Paget's osteitis deformans. The highest incidence is found between the ages of 31 and 35 years; males are more commonly affected than females. Approximately 20 per cent of all secondary chondrosarcomas occur in areas of the shoulder girdle. Although benign chondromas of the coracoid process are rare, when present they tend to undergo malignant changes. Geschickter informs the author that he has observed 5 malignant transformations occurred in all of them.

Characteristic features of significance are the low grade malignant nature of these tumors and the tendency toward repeated recurrences after conservative, usually in adequate surgery. Occasionally it is not possible to establish the presence of a pre-existing cartilaginous tumor by radiographic or histologic studies. However, the chronicity of the clinical course pursued may be a clue to the correct diagnosis of secondary chondrosarcoma superimposed on a benign lesion. Primary chondrosarcomas exhibit a clinical course that is shorter and

more rapid but, in spite of the chronicity and the low grade malignant character of secondary chondrosarcoma, most cases end fatally.

Clinical Features. Patients with secondary chondrosarcoma usually give a history of a swelling or mild pain in the affected region of the shoulder girdle, present for many months or years before the symptoms developed which forced them to seek medical aid. Often the onset was associated with trauma or strain which did not impair function; the patient may or may not be aware of a pre-existing mass. After many months with or without cause, pain, swelling and dysfunction appear, increasing progressively in intensity.

One always should suspect malignant transformation in any pre-existing cartilaginous lesion which is increasing in size and associated with a progressive increase in pain intensity and dysfunction at the shoulder joint. Pathologic fracture may occur late in the disease.

Pathology. It is often difficult to distinguish these tumors from primary chondrosarcomas. Remnants of the pre-existing tumor may facilitate making the distinction, however, no such evidence may be demonstrable. The presence of multiple benign skeletal tumors and (in older individuals) evidence of Paget's osteitis deformans in other bones may provide information relative to the origin of the neoplasm.

Histologic study discloses that the tumor consists of proliferating connective and varying amounts of myxomatous tissues interspersed with chondral elements. Cartilaginous cells in various stages of differentiation and showing mitotic figures indicate the malignant nature of the lesion. Irregular areas of calcification and ossification may be scattered throughout the tumor. It may be impossible to distinguish a secondary from a primary chondrosarcoma from microscopic study alone. It may also be difficult to distinguish the malignant forms from the benign tumors, chondroma and osteochondroma.

Roentgenographic Features Roentgenograms may disclose the remains of a chondroma or osteochondroma from which arises an irregular granular blotchy mass. Irregular areas of increased density in the

tumor are produced by islands of calcification and ossification. The tumor may cause extensive destruction of the cortex and invade the medullary canal. It may extend peripherally involving the soft tissue. Irreg-



FIG 317 (Top, left) Female 53 years old showing a chondrosarcoma which implicates the left scapula and the upper end of the humerus. Biopsy of the tumor was done elsewhere 6 months after the tumor mass was first noted, and the diagnosis of chondrosarcoma was made. The patient was treated erroneously by irradiation (Top right) Roentgenograph of the lesion a year after treatment by irradiation (Bottom left) Patient 7 days after interthoracoscapular amputation (Bottom right) Photomicrograph ($\times 150$) of tissue obtained from the amputated extremity. Observe the pleomorphic character of the chondroblasts and the paucity of stroma.



FIG 318 Chondrosarcoma of humerus in male aged 60. Tumor and upper two-thirds of humerus were resected and fibular transplant performed (J. R. Moore.)

ular branching dense shadows giving a stippled blotched appearance are characteristic radiographic features.

Treatment and Prognosis. Early radical surgery is the treatment of choice. In most instances amputation is justified. Secondary chondrosarcoma of the upper end of the humerus and the scapula rarely may be treated successfully by resection. Coley reports a case in which a total scapulectomy was done for a chondromyxosarcoma; the patient was living 11 years after operation. Such conservative measures, however, are justifiable only in cases which permit total resection of the tumor and the affected bone and which reveal low grade malignancy on careful scrutiny of the histologic structure by a competent pathologist.

Statistics reveal that recurrences invariably follow inadequate surgery. The low grade malignancy of some of these tumors

is demonstrated by the number of recurrences which occur many years after complete removal.

The disease pursues a protracted course in these cases. Death may not occur for from 2 to 10 years following recurrences.

CHONDROMA

INCIDENCE, SITE, AGE AND SEX

Typical chondromas are central lesions derived from cartilage. They are encountered most frequently in the small bones of the hands and the feet, the ribs and the vertebrae. However, they also occur in the large bones and in the bones of the shoulder girdle. Males are more commonly affected than females, a ratio of slightly more than 2 to 1. The age ranges from 20 to 70 years; the peak of incidence in all skeletal chondromas is between 20 and 30 years.

CLINICAL FEATURES

The only symptom present in the shoulder region may be soreness and mild pain, associated with exertion. On the other hand, no disturbing manifestations may be present in quiescent lesions. Growing tumors may distort the shaft slowly, causing a uniform swelling of the upper end of the humerus. Trivial trauma may produce a fracture. Some observers noted a fracture incidence of 10 per cent for all cases of central chondromas. Fracture may be the first sign indicative of the tumor. The average duration of symptoms prior to the time when the patient seeks medical advice seems to be 40.5 months.

According to some workers, malignant alterations in the tumors occur in 25 per cent of the cases. Aggravation of existing symptoms and the presence of a tumor mass which increases slowly in size is highly suspicious clinical evidence of malignant transformation.

ROENTGENOGRAPHIC FEATURES

The significant features of a central chondroma are a radiolucent area, fusiform expansion of the shaft of the bone, marked

thinning of the cortex complete lack of re active new bone formation and an occasional perforation of the cortex. The lesion is extremely rare in long bones such as the humerus but when present it closely resembles bone cyst, giant cell tumor or osteolytic osteogenic sarcoma. The diagnosis by clinical and radiographic means is extremely difficult hence histologic examination of the tissue should be made before treatment is instituted. Irregular lines of calcification within the tumor occasionally give it a multilocular appearance.

TREATMENT

In the region of the shoulder resection or total excision of the tumor is the treatment of choice. If large portions of the shaft are affected segmental resection followed by bone transplantation gives satisfactory results (Fig. 357). Large tumors of the scapula may be treated by partial or total scapulectomy. Lesions of the clavicle are best treated by resection. In rare cases resection or excision of the mass may not be possible because of size or location. In these instances amputation offers the only hope for the patient particularly if there are indications of malignant changes.

Some workers raise the question as to whether one is justified in such instances even to wait for evidence of malignant transformation believing that these cases are potentially malignant. Thus occasionally amputation may be justified before sarcomatous changes manifest themselves.

Small central chondromas may be curetted the cavity cauterized by a saturated solution of zinc chloride followed by thorough irrigation with normal saline solution. To enhance new bone formation the cavity may be packed with bone chips or bone grafts.

Any surgery performed on chondromas especially of the long bones must be definitive. If a biopsy is done complete removal of the tumor by excision or resection should follow. The marked tendency for recurrences following surgical procedures makes

such precautions imperative. Some investigators estimate the incidence of recurrences in large bones to be as high as 25 per cent.

In general lesions of such long bones as the humerus should be considered potentially malignant and treated accordingly. Lesions in the small bones of the hands and the feet are definitely benign. In the latter group extirpation of the lesion invariably produces a cure.

OSTEOCHONDROMA

INTRODUCTION

This group of benign tumors comprise the most common neoplasms of bone. While chondromas are rarely encountered in tubular bones osteochondromas are observed frequently in the ends of long bones. Osteochondromas may occur as single or multiple lesions the latter group is recognized clinically as hereditary deforming dyschondroplasia and is associated with skeletal deformities such as bending of the bones shortening resulting from interferences with normal bone growth and widening and distortion of the metaphyseal regions. Essentially these tumors all arise from cartilage the amount present being responsible for the clinical and radiographic features.

These tumors are generally conceded to be congenital lesions in many instances a familial tendency can be traced through several generations. This is especially true of hereditary deforming dyschondroplasia usually observed in the white race. Negroes are rarely affected. The familial characteristics of such tumors have been emphasized by Ollier, Hale, Jansen, Ehrenfried, the Vanzants and many others.

ETIOLOGY

The true histogenesis of these lesions is still a matter of controversy. Although their congenital nature is recognized the exact mode of their development is obscure. Geschickter and Copeland state that histogenetically osteochondromas are considered to be an exaggeration of a normal bony protuberance intended for the anchor

ing of an important tendon. At such a juncture nature normally provides a protuberance of bone bulging through a gap in the periosteum to meet an adjoining tendon which co-operates in the formation of the attachment by cartilaginous ossification within the substance of the tendon.¹ They state that in multiple exostosis "tags of perichondrium in the tendon ends proliferate to form cartilaginous and bony outgrowths. Disturbances and deficiencies in the periosteum in the metaphyseal regions lead to widening of the metaphysis and inhibition of bone growth."²

SITE INCIDENCE AGE AND SEX

As in other tumors in long bones osteochondromas usually are located at the ends of the humerus not infrequently on the scapula and rarely arise from the clavicle. Males are affected more often than females.

¹ C. F. Geschickter and M. M. Copeland, *Tumors of the Bone*, ed. 3 Philadelphia, Lippincott 1949, p. 77.

² *Ibid.*, p. 86.

the ratio being 2 to 1. The age range is from 5 to 80 years, the highest incidence occurring between 11 and 15 years.

CLINICAL FEATURES

Many lesions are asymptomatic and are discovered only accidentally by roentgenographic examination of the affected region for reasons unrelated to the neoplasm. The patient may or may not be aware of the tumor's presence. Occasionally, some form of injury directs his attention to the neoplasm.

Quiescent lesions can exist for many years without clinical manifestations; some may never give rise to symptoms. Large tumors in the region of the upper end of the humerus may interfere with motion at the shoulder joint. In these cases palpation usually discloses a firm, painless, more-or-less symmetric mass attached to the underlying bone. The consistency of the mass depends upon the amount of cartilaginous tissue. Soft tissues are freely movable over the mass.

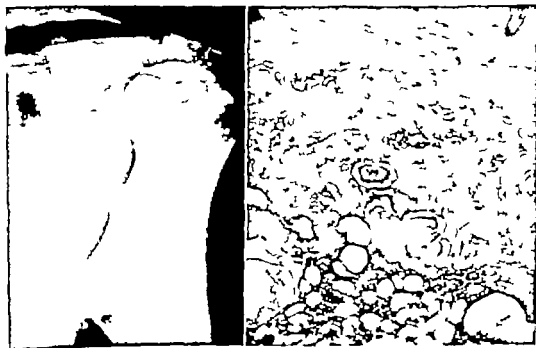


FIG. 319 (Left) Osteochondroma of upper end of the humerus of a male aged 12. Single lesion. (Right) Benign osteochondroma showing typical structural picture. The outer portion is composed of hyaline cartilage which at a lower level exhibits calcification; the inner portion or base consists of cancellous bone. The cartilaginous cap is covered with a layer of connective tissue (not seen in this section). (Photomicrograph $\times 125$)

The patient (male 12 years of age), whose shoulder is depicted in Figure 319 was unaware of the tumor but experienced some soreness and stiffness in the shoulder region following each game of baseball. The pain disappeared with rest but the patient felt numbness in the hand on several occasions following severe exertion.

The clinical manifestations are generally caused by the tumor pressing on the surrounding structures. Local paralysis may be the first indication. This was demonstrated by case J D, male 6 years of age. The mother sought medical aid because of a prominence of the right shoulder blade. Examination disclosed paralysis of the serratus anticus muscle and a large irregular osteochondroma on the ventral surface of the scapula. Skeletal survey revealed multiple bone lesions with involvement of the upper ends of both humeri (Fig. 320). Excision of the bone tumor on the scapula was followed by return of power in the affected muscle (Fig. 35).

Changes in the character of the tumors such as pain and increase in size are ominous signs. Although this group of tumors is essentially benign sarcomatous degeneration occurs more frequently than realized.

ROENTGENOGRAPHIC FEATURES

Radiographically the osteochondroma reveals rather distinctive characteristics. It may be portrayed as a bony protuberance continuous with the shaft of the humerus by a narrow pedicle or by a broad base; the latter variety constitutes the sessile form (Figs. 319 and 320). The base of the tumor is composed of normal bone; its periphery, however, is covered by a cartilaginous cap in which may be seen irregular calcified areas. Varying degrees in the size of the cartilaginous covering are discernible in different tumors. The tumor may exist as a narrow regular, imperceptible band or an enormous irregular calcifying lesion, often referred to as a "cauliflower mass." In benign lesions the periphery is sharply de-



FIG. 320 Osteochondroma of upper end of the humerus of a male aged 6. One of multiple lesions affecting all long bones and right scapula.

fined and the base shows a normal bone pattern. An irregular, hazy ill-defined periphery, a granular stippled appearance of the cartilaginous elements and irregular areas of bone resorption at the base of the tumor are suspicious indications of malignant transformations.

TREATMENT AND PROGNOSIS

Small asymptomatic tumors require no treatment. Tumors which interfere with normal function of the shoulder or press on surrounding structures sufficiently to cause pain should be removed. Excision of the mass should be complete and should be performed through normal osseous tissue beyond the limits of the tumor. Big tumors with large cartilaginous caps may not be

ing of an important tendon. At such a juncture nature normally provides a protuberance of bone bulging through a gap in the periosteum to meet an adjoining tendon which co-operates in the formation of the attachment by cartilaginous ossification within the substance of the tendon.¹ They state that in multiple exostosis "tags of perichondrium in the tendon ends proliferate to form cartilaginous and bony outgrowths. Disturbances and deficiencies in the periosteum in the metaphyseal regions lead to widening of the metaphysis and inhibition of bone growth."²

SITE, INCIDENCE, AGE AND SEX

As in other tumors in long bones, osteochondromas usually are located at the ends of the humerus, not infrequently on the scapula and rarely arise from the clavicle. Males are affected more often than females.

¹ C. F. Geschickter and M. M. Copeland, *Tumors of the Bone*, ed. 3, Philadelphia, Lippincott, 1949, p. 77.

² *Ibid.* p. 86.

the ratio being 2 to 1. The age range is from 5 to 80 years, the highest incidence occurring between 11 and 15 years.

CLINICAL FEATURES

Many lesions are asymptomatic and are discovered only accidentally by roentgenographic examination of the affected region for reasons unrelated to the neoplasm. The patient may or may not be aware of the tumor's presence. Occasionally some form of injury directs his attention to the neoplasm.

Quiescent lesions can exist for many years without clinical manifestations; some may never give rise to symptoms. Large tumors in the region of the upper end of the humerus may interfere with motion at the shoulder joint. In these cases palpation usually discloses a firm, painless, more-or-less symmetric mass attached to the underlying bone. The consistency of the mass depends upon the amount of cartilaginous tissue. Soft tissues are freely movable over the mass.

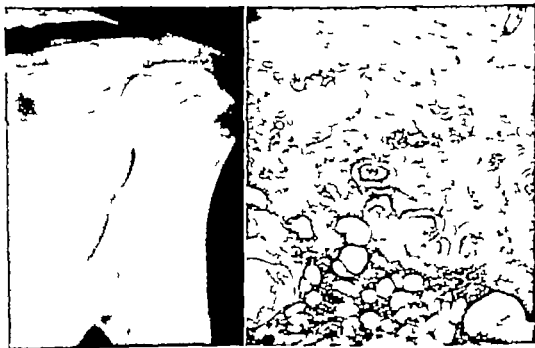


FIG. 319 (Left) Osteochondroma of upper end of the humerus of a male, aged 12. Single lesion. (Right) Benign osteochondroma showing typical structural picture. The outer portion is composed of hyaline cartilage which at a lower level exhibits calcification; the inner portion or base consists of cancellous bone. The cartilaginous cap is covered with a layer of connective tissue (not seen in this section). (Photomicrograph $\times 125$.)

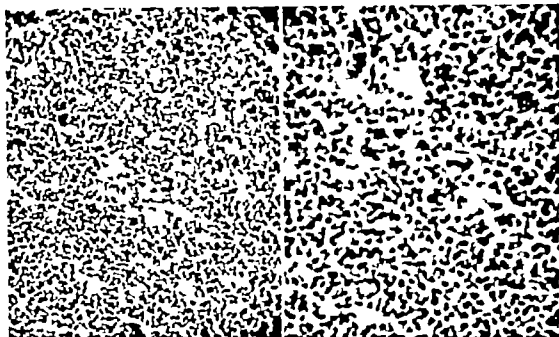


FIG 321 Plasma cell myeloma. Observe the globular eccentrically placed nuclei (Photomicrographs $\times 125$ and $\times 250$)

manifestations and of the long bones particularly the femur and the humerus are common sequelae occurring in myelomas more frequently than in any other bone tumors (in over 50 per cent of the cases). Multiple fractures are not uncommon. In widespread skeletal involvement small semifluctuant tender tumors may be palpable in the ribs, the sternum or the clavicle and the shafts of long bones may reveal uniform palpable distensions.

Systemic reactions through involvement of the blood forming elements and viscera may give rise to fever, malaise and loss of weight. Bence Jones bodies are found in over 50 per cent of the cases; these proteins have also been found in such other diseases as leukemia, carcinoma with skeletal metastases, multiple sarcoma of bone (Seegelman-Gilmore), polyfibrocystic disease (Grooves), comminuted fracture (Campbell and Horsfall) and senile osteomalacia (Rasche).

As the result of extensive destruction of the hemopoietic elements, varying degrees of secondary anemia are constant manifestations of the disease. Serum protein level may be above normal limits in many cases; occasionally it is markedly elevated.

The disease pursues a variable course



FIG 322 Multiple myeloma (plasma cell myeloma) of the shaft of the humerus. Note osteolytic nature of the lesion with no evidence of repair; also the numerous discrete punched out radiolucent areas and thinned cortex.

amenable to local excision resection of these lesions, if possible is justifiable. If histologic examination gives definite evidence of sarcomatous degeneration the lesion is treated as outlined in the treatment of secondary chondrosarcomas.

The most significant change indicative of early malignant transformation is a gradual increase in the size of the tumor. Therefore, known lesions should be checked radiographically at regular intervals to note any alteration in their physical character. Since malignant changes usually occur after 30 years of age this check up study is more essential in adults than in children.

MULTIPLE MYELOMA

INTRODUCTION

Multiple myeloma (plasma-cell myeloma) is truly an endosteal tumor. It arises from the blood forming elements of the bone marrow and it is multifocal in origin. The disease is extremely malignant and although pain may be controlled sometimes and life may be prolonged by roentgen therapy the outcome is invariably fatal. Plasma-cell myeloma is the most common of four varieties the others in order of frequency are myelocytoma, lymphocytoma and erythroblastoma. According to Coley the plasma-cell tumor constitutes 95 per cent of all myelomas.

The causative factors of the myelomas are obscure. Some investigators contend that they are not true primary bone tumors but diseases of the lymphoid system (Meyerding) or of the lymphatic hemopoietic system (Lubarsch). In the differential diagnosis several entities may be confused with this tumor: metastatic carcinoma offering the greatest difficulty. Giant-cell tumor is mentioned in the differential diagnosis when radiographic examination in the early stages shows only a single bone lesion. At times, Ewing's tumor must be considered in arriving at a diagnosis. However the age of the patient (usually over 40 years) the presence of Bence-Jones protein

in the urine (over 50 per cent of the cases) and histologic examination of the bone marrow (obtained by sternal or iliac puncture or open biopsy) suffice to establish the diagnosis in the majority of cases.

INCIDENCE, AGE AND SEX

This tumor is more common than is generally appreciated. Such improved diagnostic aids as sternal, iliac puncture and roentgenographic examination make possible the diagnosis of many lesions hitherto undiagnosed or erroneously diagnosed. The tumor is rarely encountered in those under 35 years of age usually it is found in those over 40 its highest incidence being in those between 56 and 60 years. Males are more commonly affected than females the ratio is roughly 3 to 1.

The bones which most frequently disclose clinical and radiographic manifestations of the disease are in the order of frequency the vertebrae the ribs the pelvis the femur and the humerus.

CLINICAL FEATURES

Vague pain in the lower back the abdomen and the chest may be the only complaints early in the disease. Weakness, malaise and loss of weight may be associated symptoms because of the progressive anemia. Pain may be mild at first may increase progressively in intensity and as a rule it is aggravated by exertion. Rest generally gives relief nocturnal pain not being a characteristic feature. Pain may be intermittent. In fact periods of remission which may last for months with or without treatment are common. While the interval is usually under a year cases have been recorded in which the remission lasted for 3 to 5 years.

The onset of the disease may be ushered in by a pathologic fracture (in rare cases) by the presence of a tumor or by severe pain following some form of activity. Pathologic fractures are encountered most frequently in the ribs. Fracture of vertebrae, which may be accompanied by neurologic



FIG 323 Osteoblastic metastatic foci in bones of the shoulder girdle from cancer of the breast (P Swenson Jefferson Medical College)



FIG 324 Osteolytic metastatic lesions in scapula from cancer of the breast. (H Ostrum, Philadelphia General Hospital)

evidence of bone involvement before death. It is generally known that carcinoma of certain viscera are more prone than others to metastasize to bone. Breast lesions frequently affect bone while carcinomas arising in the prostate do so less frequently. Malignancies of the bladder, the cervix, the uterus, and the gastro-intestinal tract rarely show this tendency. In 1922 Kauffmann noted on postmortem investigation that approximately two-thirds of all breast cancers and one half of all prostatic malignancies disclosed skeletal metastases.

Bones comprising the shoulder girdle are common sites for metastatic lesions. In fact renal carcinoma metastasizes to the humerus particularly to its upper third, more frequently than to any other bone of the body. Carnett and Howell (1930) reported a series of breast cancers in which the humerus and other bones of the shoulder girdle disclosed an unusually high incidence of involvement.

Secondary bone involvement may be the

first clinical manifestation of cancer elsewhere. Not infrequently the primary lesion may never be uncovered, even at autopsy.

TYPES OF METASTATIC BONE LESIONS

Secondary metastatic lesions in bone are most often multiple; however, occasionally single lesions are encountered. If single lesions are followed for sufficient time, almost always multiple involvement will be demonstrable ultimately. According to Geschickter and Copeland, single lesions occur in 25 per cent of breast cancers and they are observed more frequently in renal cancers. Although any bone in the body may be affected secondarily by any of the recognized types of cancer in general, certain cancers show a predilection for specific skeletal regions. Thyroid and renal cancers most frequently metastasize to the long bones of the extremities, particularly the humerus and the femur. Breast and prostatic tumors metastasize to the spine and the pelvis. Metastatic foci in bone may be either osteoblastic or osteolytic in nature.

As a rule, cancer cells are deposited in the

usually lasts less than two years and terminates in death

PATHOLOGY

The tumor originates from multiple foci in the cancellous spaces and the medulla and comprises a soft, gelatinous substance which varies in color (gray, brown or red), depending upon its vascularity. In the spongiosa, the points of origin grow, expand and cause resorption of the surrounding bone trabeculae and become confluent. The cortex is slowly resorbed and thinned; the periosteum is distended. Codman aptly describes the process. The result is an appearance as if the bone were blown from inside as by a series of bubbles, large and small. Characteristic of this group of tumors, there is no reactive bone formation.

Histologic examination reveals a fairly constant pattern. It consists predominantly of large round or ovoid plasma cells with globular nuclei placed eccentrically and chromatin material arranged in "spoke-like" fashion at the peripheries (Fig. 321).

ROENTGENOGRAPHIC FEATURES

Roentgenograms of multiple myeloma are fairly characteristic. As in other bones of the skeletal system, those of the shoulder girdle exhibit numerous discrete punched-out radiolucent areas of varying size. Lack of reactive new bone formation around the lesions is a prominent and striking feature. The lesions arise in a more-or-less central position; destroy all bone they come in contact with and slowly but uniformly expand the shaft (Fig. 322).

The lesion is truly an osteolytic neoplasm; hence its radiographic similarity to giant cell tumors, osteolytic sarcomas and osteolytic metastatic carcinomatous lesions. Fracture of the bone through the affected area may occur; in these cases evidence of bone healing even if present is not marked.

PROGNOSIS AND TREATMENT

The prognosis is always poor. Moderate dosages of irradiation can alleviate pain and

induce remissions. Surgery has nothing to offer except to obtain material for histologic study, either by open biopsy or aspiration. Treatment is entirely expectant in nature. In the event of fracture, protection of the humerus by splints or a plaster cast may be necessary. In fractures of the humerus, as in fractures of other tubular bones, the insertion of a Kuntscher nail in lieu of cumbersome casts, splints or traction apparatus may be justifiable to obtain fixation in these debilitated patients.

SOLITARY PLASMA-CELL MYELOMA

Such lesions have been recorded in the literature, but the author never has seen one which did not ultimately develop multiple bone involvement. Posternack and Waugh (1939) reported 30 cases from the literature and one of their own. In this series the humerus was involved in 4 cases and the clavicle in 1. It is apparent that a single lesion may offer considerable diagnostic difficulties. Its osteolytic and expansive nature may readily confuse it with giant-cell tumor and some varieties of metastatic carcinomatous lesions.

The afore-mentioned investigators noted that solitary lesions have a more favorable outlook. Seven of their cases were alive from 4 to 10 years and 6 from 7 to 10 years later. However, when considering the prognosis of a solitary myeloma, one should be cognizant of the fact that the osseous lesion is only one manifestation of a generalized affection of the hemopoietic system.

METASTATIC CARCINOMA IN BONES OF THE SHOULDER GIRDLE

INTRODUCTION

It is difficult to determine the true incidence of skeletal metastases from carcinoma. However, the incidence is considerably higher than is generally realized. Some authorities are of the opinion that roughly 50 per cent of breast, renal, prostatic and bronchiogenic carcinoma metastasize to bone and that 25 per cent reveal clinical

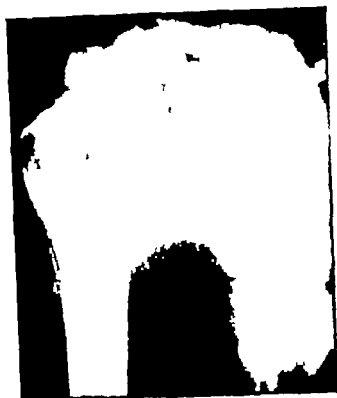


FIG 323 Osteoblastic metastatic foci in bones of the shoulder girdle from cancer of the breast (P Swenson Jefferson Medical College)



FIG 324 Osteolytic metastatic lesions in scapula from cancer of the breast. (H Ostrum, Philadelphia General Hospital)

evidence of bone involvement before death. It is generally known that carcinoma of certain viscera are more prone than others to metastasize to bone. Breast lesions frequently affect bone while carcinomas arising in the prostate do so less frequently. Malignancies of the bladder, the cervix, the uterus, and the gastro-intestinal tract rarely show this tendency. In 1922 Kauffmann noted on postmortem investigation that approximately two-thirds of all breast cancers and one-half of all prostatic malignancies disclosed skeletal metastases.

Bones comprising the shoulder girdle are common sites for metastatic lesions. In fact renal carcinoma metastasizes to the humerus particularly to its upper third, more frequently than to any other bone of the body. Carnett and Howell (1930) reported a series of breast cancers in which the humerus and other bones of the shoulder girdle disclosed an unusually high incidence of involvement.

Secondary bone involvement may be the

first clinical manifestation of cancer elsewhere. Not infrequently the primary lesion may never be uncovered, even at autopsy.

TYPES OF METASTATIC BONE LESIONS

Secondary metastatic lesions in bone are most often multiple; however, occasionally single lesions are encountered. If single lesions are followed for sufficient time, almost always multiple involvement will be demonstrable ultimately. According to Geschickter and Copeland, single lesions occur in 25 per cent of breast cancers, and they are observed more frequently in renal cancers. Although any bone in the body may be affected secondarily by any of the recognized types of cancer, in general certain cancers show a predilection for specific skeletal regions. Thyroid and renal cancers most frequently metastasize to the long bones of the extremities, particularly the humerus and the femur. Breast and prostatic tumors metastasize to the spine and the pelvis. Metastatic foci in bone may be either osteoblastic or osteolytic in nature.

As a rule, cancer cells are deposited in the

usually lasts less than two years and terminates in death

PATHOLOGY

The tumor originates from multiple foci in the cancellous spaces and the medulla and comprises a soft gelatinous substance which varies in color (gray, brown or red), depending upon its vascularity. In the spongiosa, the points of origin grow, expand and cause resorption of the surrounding bone trabeculae and become confluent. The cortex is slowly resorbed and thinned, the periosteum is distended. Codman aptly describes the process. The result is an appearance as if the bone were blown from inside as by a series of bubbles, large and small. Characteristic of this group of tumors there is no reactive bone formation.

Histologic examination reveals a fairly constant pattern. It consists predominantly of large round or ovoid plasma cells with globular nuclei placed eccentrically and chromatin material arranged in "spoke-like" fashion at the peripheries (Fig. 321).

ROENTGENOGRAPHIC FEATURES

Roentgenograms of multiple myeloma are fairly characteristic. As in other bones of the skeletal system those of the shoulder girdle exhibit numerous discrete punched out radiolucent areas of varying size. Lack of reactive new bone formation around the lesions is a prominent and striking feature. The lesions arise in a more-or-less central position, destroy all bone they come in contact with and slowly but uniformly expand the shaft (Fig. 322).

The lesion is truly an osteolytic neoplasm; hence its radiographic similarity to giant cell tumors, osteolytic sarcomas and osteolytic metastatic carcinomatous lesions. Fracture of the bone through the affected area may occur. In these cases evidence of bone healing, even if present, is not marked.

PROGNOSIS AND TREATMENT

The prognosis is always poor. Moderate dosages of irradiation can alleviate pain and

induce remissions. Surgery has nothing to offer except to obtain material for histologic study, either by open biopsy or aspiration. Treatment is entirely expectant in nature. In the event of fracture, protection of the humerus by splints or a plaster cast may be necessary. In fractures of the humerus, as in fractures of other tubular bones, the insertion of a Küntschner nail in lieu of cumbersome casts, splints or traction apparatus may be justifiable to obtain fixation in these debilitated patients.

SOLITARY PLASMA-CELL MYELOMA

Such lesions have been recorded in the literature, but the author never has seen one which did not ultimately develop multiple bone involvement. Posternack and Waugh (1939) reported 30 cases from the literature and one of their own. In this series, the humerus was involved in 4 cases and the clavicle in 1. It is apparent that a single lesion may offer considerable diagnostic difficulties. Its osteolytic and expansive nature may readily confuse it with giant-cell tumor and some varieties of metastatic carcinomatous lesions.

The afore-mentioned investigators noted that solitary lesions have a more favorable outlook. Seven of their cases were alive from 4 to 10 years and 6 from 7 to 10 years later. However, when considering the prognosis of a solitary myeloma, one should be cognizant of the fact that the osseous lesion is only one manifestation of a generalized affection of the hemopoietic system.

METASTATIC CARCINOMA IN BONES OF THE SHOULDER GIRDLE

INTRODUCTION

It is difficult to determine the true incidence of skeletal metastases from carcinoma. However, the incidence is considerably higher than is generally realized. Some authorities are of the opinion that roughly 50 per cent of breast, renal, prostatic and bronchiogenic carcinoma metastasize to bone and that 25 per cent reveal clinical



FIG 323 Osteoblastic metastatic foci in bones of the shoulder girdle from cancer of the breast. (P Swenson Jefferson Medical College.)



FIG 324 Osteolytic metastatic lesions in scapula from cancer of the breast. (H Ostrum, Philadelphia General Hospital.)

evidence of bone involvement before death. It is generally known that carcinoma of certain viscera are more prone than others to metastasize to bone. Breast lesions frequently affect bone while carcinomas arising in the prostate do so less frequently, malignancies of the bladder, the cervix, the uterus and the gastro-intestinal tract rarely show this tendency. In 1922 Kauffmann noted on postmortem investigation that approximately two-thirds of all breast cancers and one half of all prostatic malignancies disclosed skeletal metastases.

Bones comprising the shoulder girdle are common sites for metastatic lesions. In fact renal carcinoma metastasizes to the humerus particularly to its upper third more frequently than to any other bone of the body. Carnett and Howell (1930) reported a series of breast cancers in which the humerus and other bones of the shoulder girdle disclosed an unusually high incidence of involvement.

Secondary bone involvement may be the

first clinical manifestation of cancer elsewhere. Not infrequently the primary lesion may never be uncovered, even at autopsy.

TYPES OF METASTATIC BONE LESIONS

Secondary metastatic lesions in bone are most often multiple, however occasionally single lesions are encountered. If single lesions are followed for sufficient time, almost always multiple involvement will be demonstrable ultimately. According to Geschickter and Copeland, single lesions occur in 25 per cent of breast cancers and they are observed more frequently in renal cancers. Although any bone in the body may be affected secondarily by any of the recognized types of cancer, in general certain cancers show a predilection for specific skeletal regions. Thyroid and renal cancers most frequently metastasize to the long bones of the extremities, particularly the humerus and the femur. Breast and prostatic tumors metastasize to the spine and the pelvis. Metastatic foci in bone may be either osteoblastic or osteolytic in nature.

As a rule, cancer cells are deposited in the

usually lasts less than two years and terminates in death

PATHOLOGY

The tumor originates from multiple foci in the cancellous spaces and the medulla and comprises a soft gelatinous substance which varies in color (gray brown or red) depending upon its vascularity. In the spongiosa the points of origin grow expand and cause resorption of the surrounding bone trabeculae and become confluent. The cortex is slowly resorbed and thinned, the periosteum is distended. Codman aptly describes the process. The result is an appearance as if the bone were blown from inside as by a series of bubbles large and small. Characteristic of this group of tumors there is no reactive bone formation

Histologic examination reveals a fairly constant pattern. It consists predominantly of large round or ovoid plasma cells with globular nuclei placed eccentrically and chromatin material arranged in "spoke-like" fashion at the peripheries (Fig. 321)

ROENTGENOGRAPHIC FEATURES

Roentgenograms of multiple myeloma are fairly characteristic. As in other bones of the skeletal system those of the shoulder girdle exhibit numerous discrete punched out, radiolucent areas of varying size. Lack of reactive new bone formation around the lesions is a prominent and striking feature. The lesions arise in a more-or-less central position, destroy all bone they come in contact with and slowly but uniformly expand the shaft (Fig. 322)

The lesion is truly an osteolytic neoplasm hence its radiographic similarity to giant cell tumors, osteolytic sarcomas and osteolytic metastatic carcinomatous lesions. Fracture of the bone through the affected area may occur in these cases, evidence of bone healing, even if present, is not marked

PROGNOSIS AND TREATMENT

The prognosis is always poor. Moderate dosages of irradiation can alleviate pain and

induce remissions. Surgery has nothing to offer except to obtain material for histologic study, either by open biopsy or aspiration. Treatment is entirely expectant in nature. In the event of fracture, protection of the humerus by splints or a plaster cast may be necessary. In fractures of the humerus, as in fractures of other tubular bones, the insertion of a Küntscher nail in lieu of cumbersome casts, splints or traction apparatus may be justifiable to obtain fixation in these debilitated patients.

SOLITARY PLASMA CELL MYELOMA

Such lesions have been recorded in the literature, but the author never has seen one which did not ultimately develop multiple bone involvement. Posternack and Waugh (1939) reported 30 cases from the literature and one of their own. In this series the humerus was involved in 4 cases and the clavicle in 1. It is apparent that a single lesion may offer considerable diagnostic difficulties. Its osteolytic and expansive nature may readily confuse it with giant-cell tumor and some varieties of metastatic carcinomatous lesions.

The afore-mentioned investigators noted that solitary lesions have a more favorable outlook. Seven of their cases were alive from 4 to 10 years and 6 from 7 to 10 years later. However, when considering the prognosis of a solitary myeloma, one should be cognizant of the fact that the osseous lesion is only one manifestation of a generalized affection of the hemopoietic system.

METASTATIC CARCINOMA IN BONES OF THE SHOULDER GIRDLE

INTRODUCTION

It is difficult to determine the true incidence of skeletal metastases from carcinoma. However, the incidence is considerably higher than is generally realized. Some authorities are of the opinion that roughly 50 per cent of breast, renal, prostatic and bronchiogenic carcinoma metastasize to bone and that 25 per cent reveal clinical



FIG 323 Osteoblastic metastatic foci in bones of the shoulder girdle from cancer of the breast (P Swenson, Jefferson Medical College)



FIG 324 Osteolytic metastatic lesions in scapula from cancer of the breast. (H Ostrum, Philadelphia General Hospital)

evidence of bone involvement before death. It is generally known that carcinoma of certain viscera are more prone than others to metastasize to bone. Breast lesions frequently affect bone while carcinomas arising in the prostate do so less frequently. Malignancies of the bladder, the cervix, the uterus, and the gastro-intestinal tract rarely show this tendency. In 1922, Kauffmann noted on postmortem investigation that approximately two-thirds of all breast cancers and one half of all prostatic malignancies disclosed skeletal metastases.

Bones comprising the shoulder girdle are common sites for metastatic lesions. In fact, renal carcinoma metastasizes to the humerus particularly to its upper third more frequently than to any other bone of the body. Carnett and Howell (1930) reported a series of breast cancers in which the humerus and other bones of the shoulder girdle disclosed an unusually high incidence of involvement.

Secondary bone involvement may be the

first clinical manifestation of cancer elsewhere. Not infrequently the primary lesion may never be uncovered even at autopsy.

TYPES OF METASTATIC BONE LESIONS

Secondary metastatic lesions in bone are most often multiple; however, occasionally single lesions are encountered. If single lesions are followed for sufficient time, almost always multiple involvement will be demonstrable ultimately. According to Geschickter and Copeland, single lesions occur in 25 per cent of breast cancers, and they are observed more frequently in renal cancers. Although any bone in the body may be affected secondarily by any of the recognized types of cancer, in general certain cancers show a predilection for specific skeletal regions. Thyroid and renal cancers most frequently metastasize to the long bones of the extremities, particularly the humerus and the femur. Breast and prostatic tumors metastasize to the spine and the pelvis. Metastatic foci in bone may be either osteoblastic or osteolytic in nature.

As a rule, cancer cells are deposited in the

usually lasts less than two years and terminates in death

PATHOLOGY

The tumor originates from multiple foci in the cancellous spaces and the medulla and comprises a soft gelatinous substance which varies in color (gray, brown or red), depending upon its vascularity. In the spongiosa the points of origin grow, expand and cause resorption of the surrounding bone trabeculae and become confluent. The cortex is slowly resorbed and thinned; the periosteum is distended. Codman aptly describes the process. The result is an appearance as if the bone were blown from inside as by a series of bubbles, large and small. Characteristic of this group of tumors, there is no reactive bone formation.

Histologic examination reveals a fairly constant pattern. It consists predominantly of large round or ovoid plasma cells with globular nuclei placed eccentrically and chromatin material arranged in 'spoke-like' fashion at the peripheries (Fig. 321).

ROENTGENOGRAPHIC FEATURES

Roentgenograms of multiple myeloma are fairly characteristic. As in other bones of the skeletal system, those of the shoulder girdle exhibit numerous discrete punched-out radiolucent areas of varying size. Lack of reactive new bone formation around the lesions is a prominent and striking feature. The lesions arise in a more-or-less central position, destroy all bone they come in contact with and slowly but uniformly expand the shaft (Fig. 322).

The lesion is truly an osteolytic neoplasm; hence its radiographic similarity to giant cell tumors, osteolytic sarcomas and osteolytic metastatic carcinomatous lesions. Fracture of the bone through the affected area may occur, in these cases evidence of bone healing, even if present, is not marked.

PROGNOSIS AND TREATMENT

The prognosis is always poor. Moderate dosages of irradiation can alleviate pain and

induce remissions. Surgery has nothing to offer except to obtain material for histologic study, either by open biopsy or aspiration. Treatment is entirely expectant in nature. In the event of fracture, protection of the humerus by splints or a plaster cast may be necessary. In fractures of the humerus as in fractures of other tubular bones, the insertion of a Küntscher nail in lieu of cumbersome casts, splints or traction apparatus may be justifiable to obtain fixation in these debilitated patients.

SOLITARY PLASMA CELL MYELOMA

Such lesions have been recorded in the literature, but the author never has seen one which did not ultimately develop multiple bone involvement. Posternack and Waugh (1939) reported 30 cases from the literature and one of their own. In this series the humerus was involved in 4 cases and the clavicle in 1. It is apparent that a single lesion may offer considerable diagnostic difficulties. Its osteolytic and expansive nature may readily confuse it with giant-cell tumor and some varieties of metastatic carcinoma, atous lesions.

The afore-mentioned investigators noted that solitary lesions have a more favorable outlook. Seven of their cases were alive from 4 to 10 years and 6 from 7 to 10 years later. However, when considering the prognosis of a solitary myeloma, one should be cognizant of the fact that the osseous lesion is only one manifestation of a generalized affection of the hemopoietic system.

METASTATIC CARCINOMA IN BONES OF THE SHOULDER GIRDLE

INTRODUCTION

It is difficult to determine the true incidence of skeletal metastases from carcinoma. However, the incidence is considerably higher than is generally realized. Some authorities are of the opinion that roughly 50 per cent of breast, renal, prostatic and bronchogenic carcinoma metastasize to bone and that 25 per cent reveal clinical

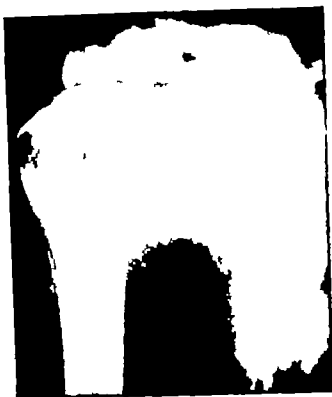


FIG 323 Osteoblastic metastatic foci in bones of the shoulder girdle from cancer of the breast. (P Swenson, Jefferson Medical College)



FIG 324 Osteolytic metastatic lesions in scapula from cancer of the breast. (H Ostrum, Philadelphia General Hospital)

evidence of bone involvement before death. It is generally known that carcinoma of certain viscera are more prone than others to metastasize to bone. Breast lesions frequently affect bone while carcinomas arising in the prostate do so less frequently. Malignancies of the bladder, the cervix, the uterus and the gastro-intestinal tract rarely show this tendency. In 1922 Kauffmann noted on postmortem investigation that approximately two-thirds of all breast cancers and one half of all prostatic malignancies disclosed skeletal metastases.

Bones comprising the shoulder girdle are common sites for metastatic lesions. In fact renal carcinoma metastasizes to the humerus, particularly to its upper third more frequently than to any other bone of the body. Carnett and Howell (1930) reported a series of breast cancers in which the humerus and other bones of the shoulder girdle disclosed an unusually high incidence of involvement.

Secondary bone involvement may be the

first clinical manifestation of cancer elsewhere. Not infrequently the primary lesion may never be uncovered, even at autopsy.

TYPES OF METASTATIC BONE LESIONS

Secondary metastatic lesions in bone are most often multiple; however, occasionally single lesions are encountered. If single lesions are followed for sufficient time almost always multiple involvement will be demonstrable ultimately. According to Geschickter and Copeland single lesions occur in 25 per cent of breast cancers and they are observed more frequently in renal cancers. Although any bone in the body may be affected secondarily by any of the recognized types of cancer, in general certain cancers show a predilection for specific skeletal regions. Thyroid and renal cancers most frequently metastasize to the long bones of the extremities, particularly the humerus and the femur. Breast and prostatic tumors metastasize to the spine and the pelvis. Metastatic foci in bone may be either osteoblastic or osteolytic in nature. As a rule cancer cells are deposited in the

of the opinion that most cases diagnosed as Ewing's tumor are actually cases of neuroblastoma

PATHOLOGIC FRACTURES

Fracture of the bones of the shoulder girdle especially the humerus is a serious complication because it adds to the difficulty in the treatment of the patient. It is a common sequela of metastatic carcinoma and multiple myeloma and may be the first indication of the disease. In fact a pathologic fracture produced spontaneously or following trivial trauma in an individual past 35 years of age is in most instances the result of metastatic carcinoma or multiple myeloma. The incidence of pathologic fracture is unusually high in metastatic renal carcinoma. Some investigators series report an incidence as high as 45.5 per cent the humerus holding second place in the order of frequency of fracture. Prostatic cancer rarely is associated with fractures; most of the metastatic lesions are characterized by new reactive bone formation and are less prone to sustain fractures.

Treatment of pathologic fracture in the bones of the shoulder girdle does not differ from pathologic fractures in other bones of the body. Intramedullary pinning, with a Küntscher nail for fracture of the humerus has been used by the author. This facilitates management of the patient, alleviates pain and does not interfere with other forms of therapy.

Spontaneous healing occurs occasionally. The amount of new bone is usually small and defective and refracture is the usual sequela. In some cases roentgen ray therapy undoubtedly destroys the tumor cells or inhibits their activity sufficiently to allow healing. Endocrine therapy and castration have been known to produce rapid bone regeneration and tumor regression in many instances of breast and prostatic tumors.

ROENTGENOGRAPHIC FEATURES

Multiple skeletal lesions in an individual past 35 years are usually indicative of

metastatic carcinoma or multiple plasma cell myeloma. The latter lesion is portrayed radiographically by multiple discrete punched out radiolucent areas without evidence of new bone formation. Solitary plasma cell myeloma may cause considerable expansion of the humeral shaft resembling osteolytic metastatic carcinoma. Diagnosis can be established by histologic study of the local tumor tissue and of the bone marrow obtained by sternal or iliac puncture.

Osteolytic solitary metastatic carcinoma lesions of the upper end of the humerus especially of renal or thyroid origin may be confused with osteolytic osteogenic sarcoma, malignant giant cell tumors or a latent bone cyst. Osteoblastic lesions especially those of prostatic cancer may closely resemble osteoblastic osteogenic sarcoma. It is obvious that histologic study of the tumor tissue is essential to establish a correct diagnosis. Blood studies revealing an elevated acid phosphatase level point to prostatic cancer.

MYOSITIS OSSIFICANS CIRCUMSCRIPTA

INTRODUCTION

This variety of myositis ossificans follows single or repeated trauma. Although the brachialis anticus and quadriceps femoris muscles are most frequently affected the lesion is not uncommon in the deltoid muscle. The fact that this lesion has often been confused with periosteal osteogenic sarcoma and (through this erroneous diagnosis occasionally has been treated by amputation) makes it imperative that the lesion be considered in this chapter dealing with bone neoplasms in the region of the shoulder joint. Myositis ossificans (especially in the early stages) may simulate osteogenic sarcoma so closely in rare instances that both surgeon and pathologist may not be able to establish a correct diagnosis in spite of all the clinical, radiographic and histologic evidence before them.

Cases are on record however in which



FIG 325 Metastatic lesion to the upper end of the shaft of the humerus from cancer of the kidney. The lesion is osteolytic in nature has caused marked expansion of the shaft and produced a pathological fracture. It must be distinguished from the osteolytic form of osteogenic sarcoma. (H. Ostrum.)

cancellous bone of the ends of long bones and in the region of the nutrient artery. Hyperplasia of the tumor cells may stimulate varying amounts of reactive new bone formation from the endosteal and periosteal elements. Cancers producing such a response are referred to as osteoblastic tumors. The involved bone areas reveal irregular areas of increased density and irregular thickening of the cortex; also they may exhibit periosteal new bone above and below the lesion (Fig 323). On the other hand, some skeletal metastatic lesions produce no reactive new bone formation, the tumor being purely a destructive lesion. Starting from a central position, it first destroys the spon-



FIG 326 Metastatic foci to the scapula from carcinoma of the prostate gland. Observe the osteoblastic nature of the tumor. (H. Ostrum.)

giosa then erodes the cortex, expands to the shaft and perforates the periosteum (Fig. 324).

These lesions comprise the osteolytic group. They are considered to grow more rapidly to attain greater dimensions than osteoblastic tumors and to be more frequently associated with pathologic fractures (Fig 325). Metastatic tumors of the prostate are most often osteoblastic (Fig 326). However, some prostatic cancers may be osteolytic; others may exhibit both characteristics. A goodly number of breast cancers also exhibit osteoblastic tendencies, most of them being osteolytic. Renal and thyroid cancers generally give rise to osteolytic metastatic lesions.

Ewing's tumor and neuroblastoma of adrenal or sympathetic origin should be mentioned in discussing types of metastatic lesions. These tumors are the source of metastatic skeletal lesions, essentially osteolytic in nature, especially in the tubular bones. While the age period in which they occur and their histologic features usually distinguish them readily from metastatic carcinoma, it may be exceedingly difficult to differentiate one from the other. Willis is

BIBLIOGRAPHY

- Albright F Note on the management of hypoparathyroidism with dihydrotachysterol J.A.M.A. 112 2592 2593 1939
- Albright F Butler A M Hampton A C., and Smith, P Syndrome characterized by osteitis fibrosa diseminata areas of pigmentation and endocrine dysfunction with precocious puberty in females Report of five cases New England J Med. 216 127 1937
- Albright F Smith P H., and Richardson A M Postmenopausal osteoporosis J.A.M.A. 116 2465-2474 1941
- Albright F Sulkowitch, H W and Bloomberg E. Comparison of effects of vitamin D dihydrotachysterol (A.T. 10) and parathyroid extract on disordered metabolism of rickets J Clin Investigation 18 165 169 1939
- Aldredge R. H. Localized fibrocystic disease of bone results of treatment in one hundred and fifty two cases. J Bone & Joint Surg 24 195 1942
- Anschutz, W Ueber einige seltene formen der knochenatrophie und osteomalacie Mitt. a.d. Grenzgeb d. Med. u. Chir 9 381 1902
- Ashurst A. P C Multiple cartilaginous exostoses (hereditary deforming chondrodysplasia) Ann. Surg 63 167, 1915
- Bancroft F W and Beal J M Healing of pathologic fractures through metastases from carcinoma of the breast Am. J Surg 69 236 1945
- Bayrd, E D and Heck F J Multiple myeloma J.A.M.A. 133 147 1947
- Bence Jones Henry On a new substance occurring in the urine of a patient with mollities ossium Part I. 1848 pp 55-62
- Bence-Jones H. Philosoph. Tr Roy Soc London, 1 55 1848
- Bennett C B Notes on early bone cyst Arch. Surg 46 608 1943
- Bennett G E. and Berkhelmer G A. Malignant degeneration in a case of multiple benign exostoses with a brief review of the literature Surgery 10 181 1941
- Bierning W L. and Albert H Secondary manifestations of hypernephromata J.A.M.A. 43 234 1904
- Busgard, J D Experimental giant cell tumor and cartilaginous exostoses of bone Arch. Surg 35 854 1937
- Bloodgood, J C Benign bone cysts, osteitis fibrosa giant cell sarcoma and bone aneurysm of the long pipe bones a clinical and pathological study with the conclusion that conservative treatment is justifiable Ann. Surg 52 145 1910
- Bloodgood J C Bone tumors myxoma central and periosteal Ann Surg 72 112 1920
- Bloodgood J C The diagnosis and treatment of benign and malignant tumors of bones J Radiology 1 147 1920
- Bloodgood J C Bone tumors benign and malignant a brief summary of the salient features based upon the study of some three hundred and seventy cases Am J Surg 34 229 1920
- Bodansky A and Jaffe H L. Phosphatase studies III serum phosphatase in diseases of the bone interpretation and significance Arch. Int Med 54 88-110 1934
- Borak J Biology of bone metastases Radiology 33 203 1939
- Borak J Relationship between the clinical and roentgenological findings in bone metastases Surg Gynec & Obst 75 599 1942
- Botterell E. H., and King E. J Phosphatase in fractures Lancet 228 1267 12 0 1935
- Brickner W M., and Milch H Pathological fractures of the humerus due to carcinoma metastases from a "silent" growth in the oesophagus Internat Clin. 1 207 1926
- Butler F E and Wooley I M Myositis ossificans Radiology 26 236 1936
- Campbell W C., and Hamilton, J F Gradation of Ewing's tumor (endothelial myeloma) J Bone & Joint Surg 23 869 1941
- Carnett J B., and Howell, J C Bone metastases in cancer of the breast, Ann. Surg 91 811 1930
- Christian S L. and Palmer L. A. An apparent recovery from multiple sarcoma Mill. Surgeon 61 42 1927 Am. J Surg 4 188 1928
- Codman E. A Nomenclature used by registry of bone sarcoma Am. J Roentgenol. 13 105 1925
- Codman E. A. Epiphyseal chondromatous giant cell tumors of the upper end of the humerus Surg Gynec & Obst. 52 543 1931
- Codman E. A Symposium on the treatment of primary malignant bone tumors Am J Surg 27 3 1935
- Codman E. A. Treatment of giant cell tumors about knee Surg., Gynec & Obst. 64 485 1937
- Coley B L. Osteogenic tumors of scapula, Am. J Surg 35 471 1937
- Coley B L. Conservative surgery in tumors of bone South. Surgeon 10 379 1941
- Coley B L. Tumors of bones and joints in Bancroft F W and Murray C R. Surgical Treatment of the Motor Skeletal System Philadelphia Lippincott 1945 p 349

sarcomatous changes occurred in areas of myositis ossificans many years after the initial trauma. Pack and Braund reported two cases in which both patients died of pulmonary metastasis. Shipley recorded 4 cases Geschickter and Copeland 2 (one of which was in the deltoid muscle) Coley has observed 1 unquestionable case

AGE, SEX AND SITE

The brachialis anticus the quadriceps femoris and the deltoid muscles constitute the site of predilection. It is seldom observed in very young children or in the aged The highest incidence occurs between 15 and 20 years but is fairly common up to 40 The highest incidence apparently coincides with the period in life in which most strenuous bodily exertion takes place Injuries sustained in football and soccer are frequently the inciting traumas Males are far more frequently affected than females.

CLINICAL FEATURES

There is always a definite history of rather severe trauma followed by tumefaction of a localized area in the deltoid muscle The mass is at first tender but not very prominent Later it slowly increases in size and acquires a hard bony consistency At this stage pain is not a significant feature and recession of the tumor may occur It rarely disappears completely

ROENTGENOGRAPHIC FEATURES

Roentgenograms immediately following an injury fail to reveal any significant findings Within 2 to 4 weeks evidence of calcification becomes apparent in the muscle If a subperiosteal hemorrhage is present the above process is also visible along the shaft of the humerus Ferguson is of the opinion

that trauma in soft tissues is responsible for stagnation of body fluids and calcification at first diffuse and amorphous, later more sharply defined dense and discrete The deposit develops an osseous pattern if subjected to functional stresses Hence, in myositis ossificans traumatica of the deltoid the calcareous mass is "distributed axially" and extends along the muscle fibers parallel with the humeral shaft It becomes more sharply defined as an osseous texture forms Generally in old cases the osseous structure is well laminated regular in outline and is apart from the shaft of the humerus lying entirely within the deltoid muscle In rare instances it may be attached to the bone for varying distances and its outline may be irregular and fuzzy Such cases may cause considerable concern in attempting to arrive at a correct diagnosis.

Early cases may challenge the most experienced investigators but late cases rarely present difficulty especially in the light of a history of severe injury When in doubt a period of waiting is justifiable before any therapy is instituted Repeated roentgenographic examinations at frequent intervals will reveal the nature of the lesion in 3 to 6 weeks which—together with the features of the clinical course pursued within this period—will aid in establishing a diagnosis.

TREATMENT

No treatment except rest to the part, is indicated in unquestionable cases. Surgical intervention in the early stages of the disease is contraindicated since the entire process may be stimulated to renewed activity and tumor recurrences Excision of the mass may be performed late in the disease when all evidence of activity has subsided Open biopsy is justifiable in doubtful cases

- the tendon of the supra pinnatus muscle Arch Surg 18 1491 1929
- Harrington A W., and Kennedy A M. Bone marrow metastases and anemia in gastric cancer Lancet 1 318 1913
- Harrison R. S. Ewings bone sarcoma Brit J Radiol 7 580 1934
- Hellner H. Das Ewings Knochensarkom reticulosarkomedes Knochenmarkes Arch f klin Chir 183 612 1935
- Herrick J B., and Hektoen L. Myeloma report of a case M News 65 239 1894
- Hirsch, E. F. and Ryerson E. W. Metastases of the bone in primary carcinoma of the lung review of so-called endotheliomas of the bones Arch. Surg 16 1 1928
- Howard, W. T., Jr and Crile G. W. Contribution to knowledge of endothelioma and perithelioma of bone Ann Surg. 42 358 1905
- Huggins C. Prostatic cancer treated by orchiectomy five years results J.A.M.A. 131 516 1948
- Jaffe H. L. Hereditary multiple exostoses Arch Path. 36 335 1943
- Jaffe H. L. Bodansky A and Blair J. E. Erzeugung von ostitis Fibrosa (osteodystrophia fibrosa) durch Epithelkörperchenextrakt Klin. Wchnschr 9 932 1930
- Jaffe H. L. and Lichtenstein L. Benign chondroblastoma of bone, Am. J Path. 18 969 1942
- Jaffe H. L. and Lichtenstein, L. Solitary unicameral bone cyst with emphasis on the x ray picture pathology and pathogenesis Arch. Surg 44 1004 1942
- Jansen M. Dissociation of Bone Growth (Exostoses and Enchondromata) or Olliers Dyschondroplasia and Associated Phenomena, The Robert Jones Birthday Volume London Oxford Univ. Press 1928 pp. 43-72
- Jones, H. T. Loose body formation in synovial osteochondromatosis with special reference to etiology and pathology J Bone & Joint Surg 6 407 458 1924
- Kauffmann E. Sekundäre geschwulste der knochen Lehrbuch der speziellen pathologischen Anatomie für Studierende und Aerzte Leipzig de Gruyter 1922 p 954
- Kienboeck, A. Ein Fall von monartikuläre Skelett chondromatose Rontgenpraxis 3 406 1931
- Kienboeck R. On the tumorous diseases of the bones primary metastatic Brit J Radiol. 31 374 1926
- Kienboeck R. Roentgendignose der Knochen und Gelenkrankheiten, Berlin Urban, 1933-1941
- King E. S. J. An example of benign osteogenic sarcoma, Brit J Surg 19 330 1931 32
- Kirklin B. R. and Weber H. M. A roentgenologic consideration of endothelial myeloma Am J Roentgenol 21 355 1929
- Kolodny A. A. A case of primary multiple endothelioma of bone with special emphasis on its roentgenologic features Arch Surg 9 636 1924
- Kolodny A. A. Angio-endothelioma of bone Arch Surg 12 354 1926
- Kolodny A. A. Bone sarcoma Surg Gynec. & Obst 44 1 163 1921
- Lang F. J. Über Knochenzysten Zentralbl f Chir 58 1618 1931
- Lewis D. Myositis ossificans J.A.M.A. 80 1231 1923
- Lichtenstein L., and Jaffe H. L. Fibrous dysplasia of bone a condition affecting one several or many bones the graver cases of which may present abnormal pigmentation of skin premature sexual development hyperthyroidism or still other extra skeletal abnormalities Arch. Path. 33 111 1942
- Lichtenstein L. and Jaffe H. L. Chondrosarcoma of bone Am J Path. 19 553 1943
- Lichtenstein L. and Jaffe H. L. Multiple myeloma Arch. Path 44 20, 1947
- Lichtenstein L. and Jaffe H. L. Ewings sarcoma of bone Am J Path. 23 43 1941
- Lubarsch O. Zur Myelomfrage Virchows Arch. f path. Anat 194 213 1906
- Macewen W. The Growth of Bone Observations on Osteogenesis An Experimental Inquiry into the Development and Reproduction of Diaphyseal Bone Glasgow Maclehose 1912
- MacGuire C. J., and McWhorter J. E. Sarcoma of bone an analysis of fifty cases Arch. Surg 9 545 1924
- MacIntyre W. Case of mollities and fragilitas ossium accompanied with urine strongly charged with animal matter Med. Chir Tr London 33 211 1850
- Martin H. E. and Ellis E. B. Biopsy by needle puncture and aspiration Ann Surg 92 169 1930
- Martin H. E., and Ellis E. B. Aspiration biopsy Surg., Gynec. & Obst. 59 518 1934
- Meyer A. R. Ein Fall von Ewingsarkom bei einen 1½ jährige Kinde Acta paediat 17 142 1934
- Meyer W. Notes on cancer with special reference to the parasitic theory J Cancer Research 8 45 1924
- Meyerding H. W. Cystic and fibrocystic disease of long bones Am J Orthop. Surg 16 253 1918
- Myerding H. W. Multiple myeloma Radiology 5 132 1925

- Coley B L. and Higinbotham N L. Solitary bone cyst *Ann Surg* 99 432 1934
- Coley B L., and Sharp G S. Primary tumors of the os calcis *Am J Cancer* 16 1053 1932
- Coley B L. Sharp, G S., and Ellis E. B. Diagnosis of bone tumors by aspiration *Am J Surg* 13 215 1931
- Coley W B. Endothelial myeloma or Ewing's sarcoma *Radiology* 16 627 1931
- Coley W B. Endothelial myeloma or Ewing's sarcoma *Am J Surg* 27 7 1935
- Coley W B. Myositis ossificans traumatica *Ann Surg* 57 305 1913
- Coley W B. and Coley B L. Primary malignant tumors of the long bones *Arch. Surg* 13 7/9 1926 14 63 1927
- Colville H C., and Willis R A. Neuroblastoma metastases in bones with criticism of Ewing's endothelioma *Am J Path.* 9 421 1933
- Connor C L. Endothelial myeloma Ewing report of fifty four cases *Arch. Surg* 12 789 1926
- Copeland, M M. and Geschickter C F. Ewing's sarcoma the nature of Ewing's tumor *Arch Surg* 20 421 1930
- Cornil, A. and Coudray P. Quelques observations des chondromes et d'osteocondromes *Rev. de chir* 38 213 1908
- Daganello U. Ein Fall von Chondro-Sarkom der Scapula *Arch. g. path. Anat* 158 265 1902
- Dahl B. Relation between Chondrodysplasia, multiple chondromatosis and Ollier's disease *Acta orthop Scandinav.* 1 12 1930
- DeSanto D A. Ewing's tumor (primary intra cortical and subperiosteal lymphangioendothelioma) *Arch. Surg* 28 66 1934
- Dresser R. Metastatic manifestations of hypernephroma in bone *Am J Roentgenol* 13 342 1925
- Ehrenfried, A. Hereditary deforming chondrodysplasia multiple cartilaginous exostoses a review of the American literature and report of twelve cases *J.A.M.A.* 68 502 1917
- Ewing J. Diffuse endothelioma of bone *Proc New York Path. Soc* n s 21 17 1921
- Ewing J. A review and classification of bone sarcoma *Arch Surg* 4 485 1922
- Ewing J. Further report on endothelial myeloma of bone *Proc New York Path. Soc* n s 24 93 1924
- Ewing J. The relation of trauma to malignant tumors *Am J Surg* 40 30 1926
- Ewing J. The classification and treatment of bone sarcoma *British Empire Cancer Campaign, London, Simpkin & Marshall, 1928*
- Ewing J. The place of biopsy in bone sarcoma *Am J Surg* 27 26 1935
- Ewing J. The modern attitude toward traumatic cancer *Bull. New York Acad. Med.* 11 281 1935
- Ewing J. A review of the classification of bone tumors *Surg. Gynec. & Obst.* 68 971 1939
- Ewing J. *Neoplastic Diseases* ed. 4 Philadelphia Saunders 1940 pp. 62 74
- Foot N C. Report of a case of malignant endothelioma with necropsy *J.M Res* 44 41/ 1924
- Foot F W Jr. and Anderson H R. Histogenesis of Ewing's tumor *Am. J. Path.* 17 497 1941
- Geschickter C F. Multiple myeloma a single lesion *Ann. Surg* 91 425 1930
- Geschickter C F. Recurrent and so-called metastatic giant cell tumor *Arch. Surg.* 20 715 1930
- Geschickter C F. *Diseases of the breast* ed. 2 Lippincott Philadelphia, 1945 p. 472
- Geschickter C F. and Copeland, M M. Multiple myeloma *Arch. Surg.* 16 80/ 1928
- Geschickter C F. and Copeland, M M. Ewing's sarcoma small round cell sarcoma of bone, *Arch. Surg.* 20 246 1930
- Geschickter C F., and Copeland, M M. *Tumors of Bone* ed. 2 Philadelphia, Am. J. Cancer 1936
- Geschickter C F., and Mastenitz, I H. Myositis ossificans *J. Bone & Joint Surg.* 20 661 1938
- Ghormley R. K., and Pollock, G A. Multiple myeloma *Surg., Gynec. & Obst.* 69 648 1939
- Ghormley R. K., Pollock G A., Hall, B E., and Beizer L. H. Multiple myeloma, *Surg., Gynec. & Obst* 74 242 1942
- Gibson A. and Bloodgood J C. Metastatic hypernephroma with special reference to bone metastasis *Surg., Gynec. & Obst.* 37 490 1923
- Gross, R. E. and Vaughn, W W. Plasma cell myeloma report of two cases with unusual survivals of six and ten years *Am. J. Roentgenol.* 39 344 1938
- Gutman A B. Tyson T L. and Gutman, E. B. Serum calcium inorganic phosphorus, and phosphatase activity in hyperparathyroidism. Paget's disease multiple myeloma and neoplastic disease *Arch. Int. Med.* 57 379-413 1936
- Hale K. Hereditary deforming chondrodysplasia or multiple exostoses report of a father and two daughters showing similar multiple symmetrical exostoses and fifty other cases collected from the English literature since 1917 *Ann Surg* 92 92 1930
- Hamilton, J E. Ewing's sarcoma endothelioma myeloma *Arch. Surg* 41 29 1940
- Harbin R M. Deposition of calcium salts in

- Stout A. P. A discussion of the pathology and histogenesis of Ewing's tumor of bone marrow. *Am J Roentgenol* 50 334 1943
- Stout A. P. Malignant tumor of lipoblasts. *Ann Surg* 119 86 1944
- Swenson P. C. The roentgenologic aspects of Ewing's tumor of bone marrow. *Am J Roentgenol* 43 204 1940
- Swift W. E., and Hallock H. Treatment of localized fibrocystic cavities of bone with curettage and packing with bone chips. *J Bone & Joint Surg* 20 411 1938
- Tavernier L. Une forme de lésion osseuse intermédiaire entre les tumeurs à myéloploïdes et les kystes des os. *Bull et mem Soc nat de chir* 52 1, 1926
- Tavernier L. Les bases du pronostic dans les tumeurs des os. *Rev med. frse* 15 287 1934
- Tavernier L. and LeClere G. Le plasmocytome solitaire des os tumeur de malignité atténuée. *Praxis* 31 3 6 1942
- Thomas A. Vascular tumors of bone. Pathological and clinical study of twenty seven cases. *Surg Gynec. & Obst.* 74 77 1942
- Wallgren, A. Myeloma, *Uppsala Läkarf. förh.* 25 113 1920
- Warren, S. L. Preliminary study of the effect of artificial fever upon hopeless tumor cases. *Am J Roentgenol* 33 75 1935
- Wilkins, W. E., and Regen E. Course of phosphatase activity in healing of fractured bone, *Proc. Soc. Exper Biol. & Med* 32 1373-1376 1935
- Wilkins W. E. Regen E. M. and Carpenter G. K. Phosphatase studies on biopsy tissue in progressive myositis ossificans. *Am J Dis Child* 49 1219 1935
- Willis R. A. Multiple myeloma. *J Coll Surg Australasia* 3 295 1930
- Willis R. A. Metastatic neuroblastoma in bone presenting Ewing syndrome with discussion of "Ewing's sarcoma." *Am J Path* 16 31, 1940
- Woodward H. Q. Acid and alkaline glycerophosphatase in tissue and serum. *Cancer Research* 2 497 503 1942
- Woodward H. Q. and Craver L. F. Serum phosphatase in the lymphomatoid diseases. *J Clin. Investigation* 19 17 1940
- Woodward H. Q. and Higinbotham N. L. The correlation between serum phosphatase and roentgenographic type in bone disease. *Am. J Cancer* 31 221 23, 1937
- Woodward, H. Q. Twombly G. H. and Coley B. L. A study of the serum phosphatase in bone diseases. *J Clin. Investigation* 15 193-201 1936
- Vanzant B. T. and Vanzant F. R. Hereditary deforming chondrodysplasia. *J.A.M.A.* 119 186 1942
- Virchow R. Ueber die Bildung von Knochen cysten. *Monatsber. d. Kgl. Akad. d. Wissenschaften. Setzung der Physikalischen mathematischen Klasse vom 12 Juni 1846*
- Vogel, P., Erf L. A. and Rosenthal, N. Hematological observations on bone marrow obtained from sternal puncture. *Am J Clin Path.* 7 436 1937

- Meyerding H W Exostosis Radiology 8 282 1927
- Meyerding H W Five year cure in case of endothelial myeloma of left femur S Clin North America 15 1219 1935
- Meyerding H W Diagnosis and treatment of Ewing's tumor (endothelial myeloma) solitary diffuse endothelioma hemangioendothelioma Collected papers of Mayo Clinic 30 1938
- Meyerding H W., and Pollock G A. Ewing's tumor (hemangioendothelioma endothelial myeloma solitary diffuse endothelioma) problem in differential diagnosis Minnesota Med. 23 416 1940
- Meyerding H W., and Valls J E. Primary malignant tumors of bone J.A.M.A. 117 23, 1941
- Morton, J J The generalized type of osteitis fibrosa cystica Arch. Surg. 4 534 1922
- Morton J J The treatment of Ewing's sarcoma of bone in Pack G T., and Livingston E. M. Treatment of Cancer and Allied Diseases New York, Hoeber 1940 vol. 3 p 2422
- Morton J J and Duffy W C A clinical and pathological study of ten bone tumors Arch. Surg. 7 469 1923
- Oberling C Les réticulosarcomes et les réticulo-endothéliosarcomes de la moelle osseuse (sarcomes d'Ewing) Bull Assoc frse p l'étude du cancer 17 259 1928
- Oberling C and Raileanu C Nouvelles recherches sur les réticulosarcomes de la moelle osseuse (sarcomes d'Ewing) Bull Assoc frse p l'étude du cancer 21 33 1932
- Pack G T and Braund, R Development of sarcoma in myositis ossificans report of three cases J.A.M.A. 119, 6 1942
- Paget J One form of chronic inflammation of bones (osteitis deformans) Tr Royal Med. & Chir Soc London 60 37 18, 65 225 1882
- Parker F Jr and Jackson H Jr Primary reticulum cell sarcoma of bone Surg Gynec & Obst 68 45 1939
- Pasternack, J G and Waugh R L. Solitary myeloma of bone Ann Surg 110 427 1939
- Pfeiffer C Ueber die Osteitis und die genese Therapie der Knochenzysten Beitr z klin Chir 53 4/3 1907
- Phemister D B Chondrosarcoma of bone Surg Gynec. & Obst 50 216 1930
- Phemister D B Undifferentiated round cell sarcomas Ann Surg 93 125 1931
- Phemister D B Conservative surgery in the treatment of bone tumors Surg Gynec & Obst. 70 355 1940
- Phemister D B and Gordon J E The etiology of solitary bone cyst J.A.M.A. 87 1429 1926
- Piney A. Carcinoma of the bone marrow Brit. J Surg 10 235 1922
- Piney A. The relation of the bone marrow to the lymphatic system Arch Surg 13 615 1926
- Pommer G Zur Kenntnis der progressiven Hamatom und Phlegmasieveränderungen der Röhrenknochen Arch. f Orthop u. Unfall. Chir 17 17 1920
- Recklinghausen F Die fibrose oder deformierende Osteitis, die Osteomalacie und die osteoplastische Carcinose in ihren gegenseitigen Beziehungen, in Festschrift für R. Virchow zu seinem 41 Geburtstag gewidmet Berlin G Reimer 1891
- Regen E M., and Wilkins, W E. Phosphatase in heterotopic bone formation following transplantation of bladder mucosa J Lab & Clin. Med. 20 250-252 1934
- Santos J V Inflammatory nature of bone cyst J Bone & Joint Surg 12 150 1930
- Schramm, G Pathogenesis of cartilaginous exostosis and enchondromas Arch. f Orthop. 27 421 1929
- Sevier C E. Ewing's tumor J Bone & Joint Surg 12 929 1930
- Shallow T A. Raker N., and Fry K. Primary malignant tumors of bone with special reference to osteogenic sarcoma J Internat. Coll. Surgeons 6 89 1945
- Sherman R S and Snyder R E. The roentgenological appearance of primary reticulum cell sarcoma of bone Am. J Roentgenol. 58 291 1947
- Shipley A M Ossifying hematoma and allied conditions Arch. Surg 41 516 1940
- Summons C C Bone sarcoma factors influencing prognosis Surg., Gynec. & Obst 68 67 1939
- Sisk, J N. Bone tumors Radiology 13 115 1929
- Snapper I Medical Clinics on Bone Diseases A Text and Atlas New York, Interscience, 1943
- Snapper I Stilbamidine and pentamidine in multiple myeloma J.A.M.A. 133 157 1947
- Snyder R E and Coley B L. Further studies on the diagnosis of bone tumor by aspiration biopsy Surg Gynec & Obst. 80 517 1945
- Stewart F W Primary liposarcoma of bone, Am J Path. 7 87 1931
- Stewart F W Occupational and post traumatic cancer Bull. New York Acad Med. 23 145 1947
- Stewart F W and Taylor A. L. Observation on solitary plasmocytoma J Path. & Bact. 35 541 1932
- Stone R. S Angiosarcoma and myeloma, Am J Roentgenol. 22 153 1929

It is obvious therefore that surgical intervention is not indicated in the above lesions until there is definite clinical evidence of shoulder joint dysfunction resulting from extensive tears of the cuff. Impacted fractures of the upper end of the humerus do not require reduction; excellent painless motion without normal anatomic repositioning of the fragments is the rule.

However a group of lesions do demand primary surgical intervention; recognition and prompt and adequate management of these conditions will produce gratifying results. The surgeon's failure to so proceed results in marked dysfunction of the shoulder joint. This is especially true of fracture-dislocations of the humeral head, fracture of the greater tuberosity with retraction beneath the acromion and extensive tears of the musculotendinous cuff which may occur with simple dislocation or fracture-dislocations.

Accurate diagnosis cannot be made in many instances; yet exploration of the subacromial region is indicated. In other cases visualization of the affected area may disclose unsuspected pathologic disorders at great variance with what the surgeon anticipated preoperatively. It becomes apparent that the operator must be well trained and experienced in surgery of the region of the shoulder joint and be prepared to treat adequately all lesions that may be encountered.

Knowledge and proper management of certain peculiarities of this region will prevent undesirable sequelae and promote restoration of function. Immobilization of the shoulder joint favors formation of adhesions in and between the strata of soft tissues comprising the shoulder; obliteration of the gliding parts between the various strata, formation of periarticular adhesions and contracture and shortening of the muscles, particularly the adductor group. All surgeons recognize the seriousness and the disability of a stiff, painful shoulder. Therefore operative procedures should strive for early mobilization of the part which is the

best and only prophylactic measure at our disposal.

It has been pointed out that frozen shoulder is more apt to occur in individuals past 30 years of age, in those whose general economy is below par and in those who are victims of chronic debilitating diseases. Maintenance of normal motion in the joints distal to the shoulder, good tonicity of the muscles and elasticity of other soft tissues of the limb will enhance rapid restoration of function at the shoulder joint. Rigid and complete postoperative immobilization of the upper extremity must be avoided at all times unless it is an absolutely essential requirement in the plan of treatment. Active exercises and use of the extremity are far superior to any passive forms of physiotherapy.

ANATOMIC CONSIDERATIONS

PRINCIPAL GLIDING MECHANISMS

From a practical viewpoint, the muscles of the shoulder region form two sleeves; one glides within the other. The deltoid muscle is the principal component of the outer sleeve while the short rotator muscles (supraspinatus, infraspinatus, teres minor and subscapularis muscles) comprise the inner sleeve. Between the two is an efficient gliding mechanism which allows free, smooth, unrestricted movements. This mechanism consists of the subacromial bursa (see *Anatomy of Subacromial Bursa*) and loose fine areolar tissue. To insure maximum function it is essential to preserve this gliding mechanism. However as previously pointed out, part or all of the superior walls of the subacromial bursa may be removed without ill effects. There exists another gliding mechanism in this region which plays a major role in movements of the scapulohumeral motion; namely, the tendon-tendon-sheath apparatus of the long head of the biceps brachii muscle.

It will be recalled that the synovial lining of the scapulohumeral joint continues distally in the bicipital groove and is then

Surgical Approaches and Procedures

ANATOMIC CONSIDERATIONS

APPROACHES TO THE SHOULDER JOINT REGION

ARTHIRODESIS OF THE SCAPULOHUMERAL JOINT

MUSCLE TRANSPLANTATIONS

PARALYSIS OF THE DELTOID MUSCLE

TRANSPLANTATION OF FASCIAL EXTENSION OF THE TRAPEZIUS MUSCLE

TRANSPLANTATION OF THE BICEPS AND THE TRICEPS MUSCLE

TRANSPLANTATION OF THE ORIGIN OF THE POSTERIOR PORTION OF THE DELTOID

FASCIAL TRANSPLANTS IN PARALYTIC DISORDERS ABOUT THE SHOULDER GIRDLE

PARALYSIS OF THE SPINAL AND ELEVATOR

MUSCLES OF THE SCAPULA

PARALYSIS OF THE SERRATUS ANTERIOR MUSCLE

PARALYSIS OF THE RHOMBOIDEI AND LEVATOR SCAPULAE MUSCLES, WITH GOOD POWER IN THE SERRATUS ANTERIOR MUSCLE

AMPUTATIONS

RESECTIONS

GENERAL CONSIDERATIONS

The goal of all operative procedures in the region of the shoulder joint is to restore *maximum painless function* approaching the normal. Failure to attain this end results in marked impairment in the *usefulness* of the hand, a highly specialized organ which depends for optimum performance upon the integrity of the shoulder joint. Essentially the principal function of the upper extremity is to place and maintain the hand in positions necessary to execute specific duties. To achieve this goal certain prime requisites are mandatory.

All surgical procedures must be governed by an accurate knowledge of the topographic anatomy and comprehension of the functional mechanics of the structures for which surgery is contemplated. So intimately interrelated are the structures comprising the shoulder joint that dysfunction of one will be reflected in the total performance of this complex mechanism. On the other hand an appreciation of the alterations compatible with good function possible in this region will facilitate some surgical procedures and permit certain modifications of normal anatomy.

For example it has been demonstrated clinically that removal of all or portions of the acromion in no way affects the efficiency of the deltoid muscle. This fact also allows resection of the bony prominence with impunity when greater exposure of the subacromial region is desired. Also the coracoacromial ligament may be severed or resected and portions or all of the roof of the subacromial bursa excised without impairing the normal, smooth, rhythmical movements of the shoulder joint. (In Chapter 3 the functional mechanics of the shoulder joint have been considered.)

Operative procedures are not justifiable without assurance that function will be improved. Such procedures are contraindicated if their aim is primarily to restore normal anatomy without improving function. As has been shown within certain limits tears of the musculotendinous cuff are compatible with complete painless motion of the shoulder. Fresh traumatic lesions pointing to rupture of the cuff do not demand immediate exploration of the subacromial area. Many individuals so afflicted regain complete usefulness of the arm after the acute painful symptoms subside, if the tear is not too severe.

the muscle substance alternating with those passing downward. The oblique fibers arising from the descending septa insert into the ascending septa. Such a complex scheme does not exist in the anterior and posterior portions of the deltoid muscle. Between the adjacent borders of the deltoid and the pectoralis major muscles is a distinct interval which widens at its proximal end to form the inferior clavicular fossa or deltopectoral triangle.

CEPHALIC VEIN

The cephalic vein traverses the deltopectoral groove picking up tributaries as it proceeds cephalad. It occupies progressively a lower level as it ascends until it lies on the clavipectoral fascia below the clavicle; it perforates the costocoracoid fascia, passes anterior to the axillary artery and empties into the axillary vein. The cephalic vein is a landmark in surgical procedures in this region; it points to a natural plane of cleavage to the structures at a lower level. The axillary vessels are readily identified by tracing the cephalic vein upward. The origin of the deltoid muscle is of practical significance. It originates in the clavicle, the acromion and the spine of the clavicle and consists of tendinous fibers which blend with the periosteum of these bony structures. Part or all of the origin of the muscle may be detached subperiosteally and still sufficient sturdy tissue remains to effect firm reattachment of the muscle on either side of the line of division.

PECTORALIS MAJOR

The pectoralis major is the large fan-shaped muscle covering the upper and anterior portions of the thoracic cage. It is composed of muscle fibers arising from three origins. The clavicular portion takes origin from the anterior surface of the inner half of the clavicle; the sternal portion from the anterior surface of the sternum, the cartilages of the first seven ribs and the abdominal portion from the aponeurosis of the oblique externus abdominis. All fibers

are directed laterally converging, as they near the shaft of the humerus. They insert into the crest of the greater tuberosity by means of a broad flat bilaminar tendon U-shaped whose layers are continuous below but opened above. The anterior lamina is formed by the fibers of the clavicular and the upper sternal portions of the muscle; the fibers from the abdominal and lower sternal portions forming the posterior lamina which ascends to a higher level than the anterior lamina and giving off fibers—both those which span the intertubular groove and others which are continuous with the fibrous capsule of the glenohumeral joint.

BONY LANDMARKS

Four bony eminences are the landmarks for all operative procedures in this region. These are the acromion process, the greater and the lesser tuberosities and the coracoid process. The acromion process forms the tip of the shoulder and overhangs the greater and the lesser tuberosities; all are readily palpable. The coracoid process lies immediately beneath the anterior border of the deltoid muscle. It is situated in the interval formed by the clavicle and the inner aspect of the head of the humerus, one fingerbreadth below the inferior margin of the clavicle. It too is easily felt. The conjoined tendon of the short head of the biceps brachii muscle and the coracobrachialis arises from the coracoid process into which is inserted the pectoralis minor muscle.

This process is an important guide to the large vessels and nerves of the arm which pass under cover of the pectoralis minor to the inner aspect of the coracoid process thence to the inner side of the coracobrachialis. Forceful retraction of the muscles attached to the coracoid process may inflict trauma to the adjacent nerves and vessels. The author has observed several cases of severe causalgia which developed post-operatively as a result of prolonged and forceful traction on these structures.

Other bony landmarks are the spine of

reflexed proximally onto the biceps tendon. The old concept that the tendon glides in the groove has given way to the newer opinion that the humerus slides up and down the tendon during all motions at the

significance of this gliding plane is evident when it is converted into dense, plastic adhesions which bind the short rotator muscles to the capsule, a condition notable in frozen shoulder.

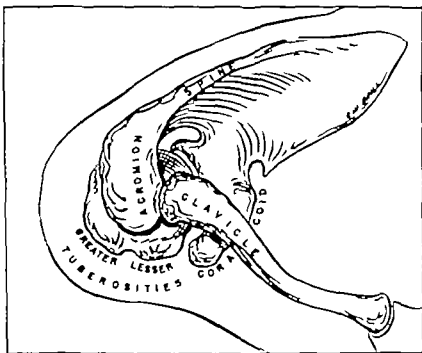


FIG 327 Bony landmarks of the shoulder girdle

scapulohumeral joint and that although the biceps tendon becomes taut during these motions it does not move within the groove. Interference with free excursion of the humeral head on the tendon results in marked impingement of elevation of the upper extremity. Yet it has been shown that total obliteration of this mechanism by anchoring the biceps tendon to the humerus or coracoid process is compatible with painless normal range of motion.

It was previously noted that the four short rotator muscles (supraspinatus, infraspinatus, teres minor, and subscapularis muscles) terminate in short stout tendons which blend with one another and with the fibrous capsule to form the musculotendinous cuff. Proximal to the line of fusion of the tendons with the capsule the muscles lie directly on the fibrous capsule; the two structures are separated only by a layer of filmy areolar tissue which permits the two strata to glide by one another with minimum friction during movements of the limb. The

DELTOID MUSCLE

The deltoid which forms the outer of the two muscular sleeves is a massive triangular muscle which drapes itself around the anterior, lateral and posterior aspects of the outer third of the clavicle, the lateral border of the acromion process and the lower border of the spine of the scapula. Its fleshy fibers converge distally to form a stout short tendon which inserts into the deltoid tubercle. The arrangement of the anterior and posterior fibers of the deltoid which arise from the clavicle and the spine of the scapula respectively differs from that of the central fibers which arise from the acromion.

The central muscle mass consists of oblique fibers which arise in a pinnate fashion from either side of 4 to 5 tendinous bands whose proximal ends are attached to the acromion. These fibrous septa proceed distally parallel with one another and become lost in the muscle substance. From the tendinous insertion of the deltoid 3 or 4 similar tendinous strands pass upward into

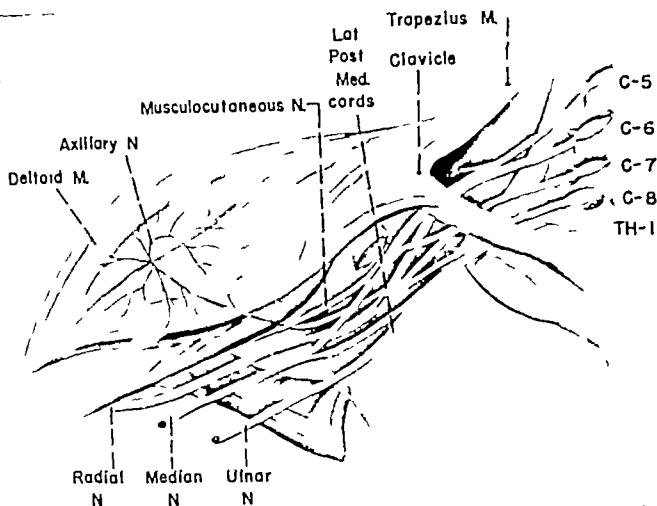


FIG 328 Course of the axillary nerve around the neck of the humerus.

the integument over the lower posterior and lateral portions of the deltoid (Figs 206 and 207)

The anterior branch together with the posterior humeral circumflex artery winds around the surgical neck of the humerus giving off muscular branches which pass vertically upward into the substance of the deltoid muscle. As it approaches the anterior border of the deltoid it diminishes progressively in size. Throughout its entire course from its origin posteriorly to its termination anteriorly the anterior division runs a horizontal course roughly 2 inches below the lateral border of the acromion process (Fig 328).

It becomes apparent that a zone of safety exists between the course of the nerve and the origin of the deltoid which can be utilized in surgical approaches to the sub-acromial region and to the glenohumeral joint. From a practical viewpoint incisions in the anterior aspect of the deltoid (from

$\frac{1}{2}$ to $\frac{3}{4}$ inches from the anterior border of the muscle) which cross the path of the axillary nerve do not in any way diminish the efficiency of the muscle. This is not true if the nerve is severed in its anterolateral, lateral or posterior position.

The suprascapular nerve is formed by fibers derived from the fifth and the sixth cervical nerves under cover of the trapezius and the omohyoid muscles. The nerve proceeds laterally and passes beneath the superior transverse scapular ligament to reach the suprascapular fossa. In this position it lies in relation with the transverse scapular artery (a branch of the thyrocervical trunk) which passes above the suprascapular ligament. Both nerve and artery then proceed beneath the supraspinatus muscle. In this position the nerve gives off branches to the supraspinatus muscle and twigs to the shoulder joint. It then winds around the spine of the scapula to the infraspinous fossa where it supplies branches to the infra

the scapula and the clavicle, both are subcutaneous and readily palpable throughout their entire lengths. The acromion and the spine of the scapula together with the clavicle form the arch of the shoulder.

Both the acromioclavicular and the sternoclavicular joints occupy very superficial positions and are readily palpated as compared with the glenohumeral joint which is situated at a much deeper level and cannot be palpated. The acromioclavicular joint is recognized by the enlarged outer end of the clavicle which articulates with and projects above the flat superior surface of the acromion. The sternoclavicular joint is identified by the enlarged sternal end of the clavicle which is readily felt immediate to the outer border of the sternal head of the sternocleidomastoid muscle.

SUBACROMIAL BURSA

Other structures of surgical significance are situated under cover of the deltoid muscle. These are the coraco-acromial ligament, the subacromial bursa, the musculotendinous cuff and the long head of the biceps brachii. These structures have been considered in detail in Chapter 2. However, attention again must be called to the topographic anatomy of the subacromial bursa. It is a concave-convex structure whose floor is firmly attached to the superior surface of the musculotendinous cuff and the greater tuberosity while its roof is adherent to the under surface of the deltoid muscle. It is prolonged under the coraco-acromial ligament and the acromion. It may extend medially as far as the base of the coracoid process; this portion is often referred to as the subcoracoid bursa.

Incisions through the anterior and lateral aspects of the deltoid muscle may open the roof of the bursa unless great care is exercised in separating it from the overlying muscle fibers. However, in order to gain access to that portion of the musculotendinous cuff beneath the acromion it will be necessary to split the superior wall of the bursa.

MUSCULOTENDINOUS CUFF

The fibers of the tendons of the short rotator muscles intermingle with the fibers of the capsule proximal to their point of insertion to form the musculotendinous cuff. This inserts into the sulcus of the head of the humerus and the greater and the lesser tuberosities. Between the subscapularis and the supraspinatus tendons is an interval occupied by the coracohumeral ligament. This break in the continuity of the cuff provides an excellent approach to the inside of the glenohumeral joint, the exposure being attained by dividing the capsule in the above interval parallel with the fibers of the coracohumeral ligament. Such an incision can be extended readily as far as the brim of the glenoid cavity.

NERVES

The topographic anatomy of the nerves supplying the structures of the shoulder joint must be constantly in the mind of surgeons working in this region. Damage to these structures results in marked dysfunction of the upper extremity. The axillary, the suprascapular and the musculocutaneous nerves are most vulnerable during operative procedures.

All surgical approaches to the shoulder joint are more or less designed around the course of the axillary nerve. Its fibers are derived from the fifth and the sixth cervical nerves which traverse the posterior cord. Posterior to the axillary artery at the lower border of the subscapularis muscle the posterior cord gives off the axillary nerve. This together with the posterior humeral circumflex artery passes backward through the quadrilateral space thence winds around the surgical neck of the humerus and under cover of the deltoid muscle reaches the anterior aspect of the shoulder. At its exit from the quadrilateral space it divides into anterior and posterior branches.

The posterior branch innervates the teres minor and the posterior portion of the deltoid. It then continues laterally as the lateral brachial cutaneous nerve supplying

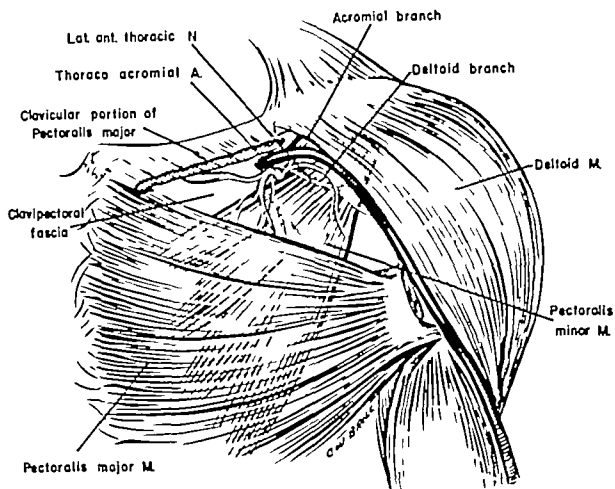


FIG. 330 The costocoracoid membrane.

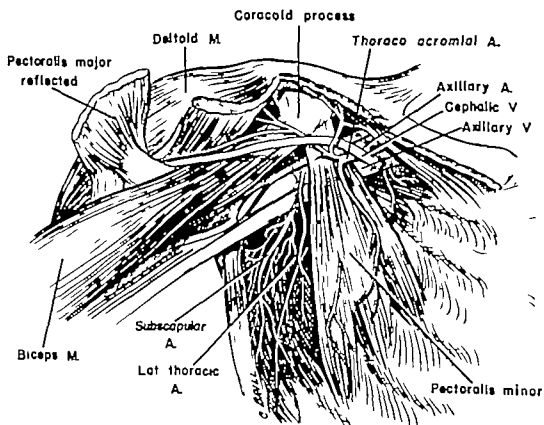


FIG. 331 Vessel under the pectoralis minor muscle.

spinatus muscle, the capsular ligaments of the shoulder joint, and the scapula (Fig 329)

The musculocutaneous nerve is composed of fibers from the fifth, sixth and seventh

and empties into the axillary vein, therefore acting as a useful guide to the large vessels the acromiothoracic artery (after passing through the fascia) gives off the pectoral, the acromial, the deltoid and the clavi-

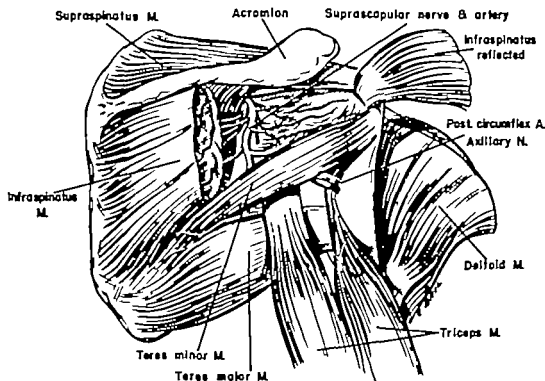


FIG 329 Course of the suprascapular nerve

cervical nerves and comes off the lateral cord below the lower margin of the pectoralis minor muscle close to its insertion into the coracoid process. It passes through the substance of the coracobrachialis muscle to the lateral aspect of the arm between the biceps brachii and the brachialis muscles. This nerve may be traumatized by surgical procedures in the anterior region of the shoulder in which traction in a medial direction is made on the structures attached to the coracoid process (Fig 209)

ARTERIES AND VEINS

The acromiothoracic artery (a branch of the second part of the axillary artery) the cephalic vein and the lateral anterior thoracic nerve are beneath the clavicular origin of the pectoralis major muscle. All pierce the costocoracoid membrane (part of the clavipectoral fascia) in their respective courses. The cephalic vein pierces the fascia

and empties into the axillary vein, therefore acting as a useful guide to the large vessels the acromiothoracic artery (after passing through the fascia) gives off the pectoral, the acromial, the deltoid and the clavi-

cular branches. When working in this region careful dissection, isolation and ligation of these vessels will eliminate profuse bleeding (Figs 330 and 331). The anterior and the posterior humeral circumflex arteries, both branches of the axillary artery, must be given due consideration in surgical procedures in this region. As previously noted, the posterior humeral circumflex is in intimate relationship with the axillary nerve throughout its entire course. It is larger than the anterior humeral circumflex artery and arises from the axillary artery at the lower border of the subscapularis muscle. Together with the axillary nerve, it proceeds backward through the quadrilateral space formed by the teres major below, the subscapularis and the teres minor above, the long head of the triceps brachii medially and the surgical neck of the humerus laterally. It passes around the neck of the humerus, anastomosing with the

shoulder with maximum efficiency and ease. The ideal position permits easy access to the anterior, the lateral, the posterior and the superior aspects of the shoulder. It maintains the patient's head in a fixed position away from the operative field, allows manipulation in all directions of the affected extremity and finally permits two assistants to work without obstructing the field. Many surgeons still utilize the supine position, with a sandbag under the shoulder. This position does not provide the helpful features enumerated. Moreover, it adds to the surgeon's difficulties by failing to provide ready access to the lateral, the superior and above all the posterior regions of the shoulder. Then too the surgeon and his assistants are forced to work in awkward and strained positions.

Of all the positions the sitting position has proved to be closest to the ideal posture. A tilting operating table is necessary. The upper part of the table beneath the patient's trunk is raised to an inclined plane of 60° to 70° from the horizontal and the lower part is broken and elevated under the knees so that both hips and knees are flexed sufficiently to prevent the patient sliding down on the table. The patient is placed so that the shoulder extends over the side of the table. By tilting the whole table slightly away from the surgeon, accessibility to all parts of the shoulder is increased. The head is turned to the opposite side and the arm is draped separately to permit manipulation of the extremity (Fig. 332). This position has proved to be most practical for all surgical procedures on the shoulder.

In cases in which the operative procedure is to be limited entirely to the anterior aspect of the shoulder, such as removal of calcareous deposits from the musculotendinous cuff or exploration of the subacromial bursa, the supine position with the shoulder over the side of the table and a sandbag under it suffices. The prone position may be used if the procedure is restricted to the posterior aspect of the shoulder. Here the outstretched arm is placed on a table or arm board, a sandbag is placed under the chest

wall on the affected side and the head is turned to the opposite side.

Draping the arm correctly so as to permit manipulation appears to be an insignificant step in preparation of the patient. Yet it detracts from the smoothness of the operation if not properly done. Otherwise the drapes may be pulled off, exposing unsterile parts, contaminating the surgeon or the assistant or both, often during manipulation of the arm. This is avoided by using two segments of sterile stockinet, closed at one end and long enough to cover the extremity from the finger tips to the middle of the upper arm. First one layer and then the second is placed over the arm and fastened by a layer of sterile gauze bandage wrapped tightly around the arm.

ANESTHESIA

Some of the simpler procedures may be performed under local anesthesia. This is especially useful when the co-operation of the patient is needed. Intravenous Pentothal Sodium when not contraindicated is a useful anesthetic agent. It is easily administered and eliminates the difficulties caused by an anesthetist struggling to give inhalation anesthesia. For cases requiring more extensive surgery, intratracheal anesthesia is by far the most desirable anesthesia and eliminates the usual difficulties encountered with routine inhalation anesthesia.

SKIN INCISION

Vertical skin incisions produce wide ugly scars which are often the source of considerable mental distress—especially among women. Unfortunately, most extensive approaches to the shoulder joint region have an anterior vertical limb running parallel with the deltopectoral groove. The smaller incisions generally used to remove calcareous deposits from the musculotendinous cuff also pursue a short vertical course. This is not true of superior incisions which parallel the normal skin creases.

Hitchcock has popularized a curved anterior skin incision which may be used instead of the anterior vertical incision. This



FIG 332 Patient in sitting position for operative procedure in the region of the shoulder



FIG 334 Transacromial incision, with extension downward of the anterior limb of the incision. This route gives the best cosmetic effect.

terminal branches of the anterior humeral circumflex artery. Throughout its course it distributes vessels to the deltoid muscle and the shoulder joint.

The anterior humeral circumflex artery arises from the axillary artery opposite the posterior. It runs laterally directly beneath the coracobrachialis and the short head of the biceps brachii to reach the anterior aspect of the surgical neck of the humerus. At the intertubercular groove it gives off an ascending branch which traverses the

groove to supply the humeral head and the shoulder joint. It proceeds thence around the neck of the humerus beneath the deltoid giving off ascending and descending branches and finally anastomosing with the posterior humeral circumflex artery.

POSITION OF PATIENT

Correct positioning of the patient is essential to perform surgical procedures on the



FIG 333 (Left) A wide, ugly scar has formed following the exposure of the anterior region of the shoulder through a deltopectoral incision. (Right) Observe the cosmetic result in the two shoulders following the use of a curved incision on the right and an anterior vertical incision on the left side.

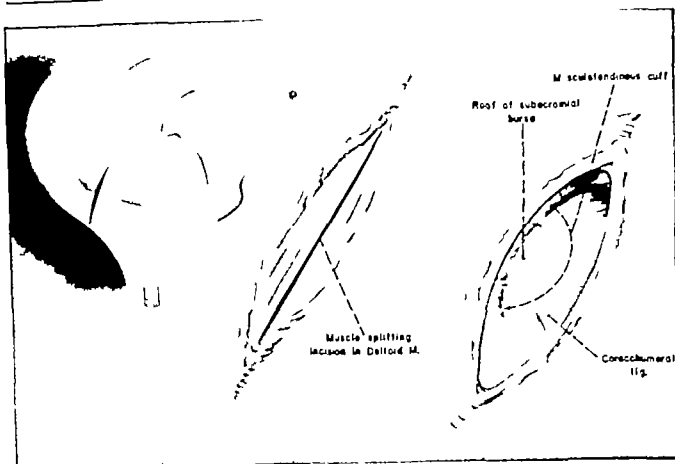


FIG. 336 Bursal incision (*Left*) Skin incision. (*Center*) Line of separation of fibers of deltoid muscle. (*Right*) Exposure of subacromial bursa and musculotendinous cuff

humeral head. The superior approaches afford better exposure of these lesions. The sitting position is preferred with the arm at the side and a small sandbag under the shoulder.

A slightly curved skin incision is made along the border of the deltoid muscle beginning at the lower border of the clavicle just to the inner side of the coracoid process and extending downward to a point just cephalad to the deltoid tuberosity of the humerus. The adjacent borders of the deltoid and the pectoralis major muscles forming the deltopectoral cleft come into view. The cephalic vein is seen transversing the cleft. To avoid injury to this vein a line of cleavage is developed in the anterior fibers of the deltoid approximately 1 centimeter from its anterior border. This medial strip of muscle fibers together with the cephalic vein and the pectoralis major muscle, is retracted medially and the deltoid laterally. While splitting the deltoid muscle numer-

ous small veins and the deltoid branch of the thoraco-acromial artery are encountered. These are ligated to ensure a bloodless field.

The following structures are now visualized. The coracoid process to which are attached the pectoralis minor, the coracobrachialis and the short head of the biceps brachii muscles, the intertubercular sulcus, the long head of the biceps brachii muscle, the inner portion of the subacromial bursa and its subcoracoid extension and the anterior aspect of the greater tuberosity. External rotation of the extremity brings into view the tendon of the subscapularis muscle as it inserts into the lesser tuberosity. Separation of the long and the short heads of the biceps brachii exposes the tendons of the teres major and the latissimus dorsi muscles. The former inserts into the crest of the lesser tuberosity and the latter into the bottom of the intertubercular groove. The tendon of the teres major occupies a

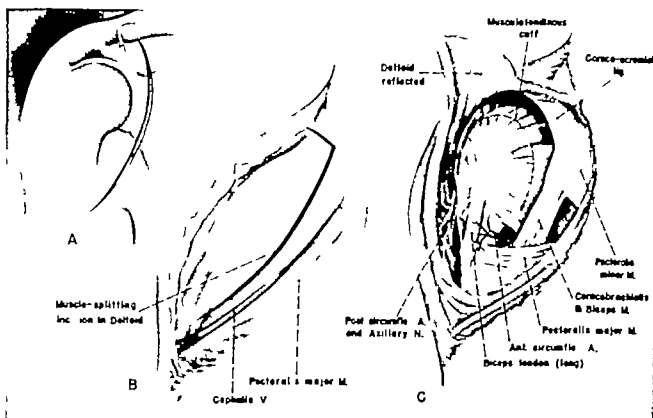


FIG. 335 Deltopectoral incision. (A) Skin incision. This may be replaced by an S-shaped incision. (B) Line of cleavage in anterior fibers of the deltoid muscle. (C) Exposures of structures beneath the coracoacromial arch.

produces a better cosmetic result and minimum scarring and helps to prevent the spreading of the incision. In exposures utilizing both a horizontal and a vertical limb as the incision of Cubbins et al. and Henry, the writer substitutes the curved incision of Hitchcock for the anterior limb (Figs. 333 and 334).

APPROACHES TO SHOULDER JOINT REGION

ANTERIOR APPROACHES

All anterior incisions are designed to protect the axillary nerve from injury. The course of the nerve must be constantly in the surgeon's mind while making one of these incisions. As already noted, the axillary nerve runs a horizontal course under cover of the deltoid about 2 to 2½ inches below the lateral margin of the acromion process. On the skin surface, this line lies slightly above a point midway between the

tip of the acromion and the deltoid tuberosity on the humerus. Thus, it is apparent that a "safety zone" exists proximal to the course of the nerve and the deltoid fibers within this zone may be split without fear of injury to the nerve. From a practical viewpoint, an incision in this zone may extend from the margin of the acromion downward for 4 centimeters without traumatizing the nerve. Beyond this, as pointed out by McLaughlin, the risk of inflicting damage to the nerve increases with each centimeter that the incision is extended downward. Division of the nerve results in denervation of the muscle fibers anterior to the site of severance (Fig. 328).

Approach through the Deltopectoral Cleft. This is a common approach to the anterior aspect of the shoulder region providing excellent exposure of lesions of the long head of the biceps brachii muscle and the bicipital groove. It may be used for fracture and fracture-dislocations of the

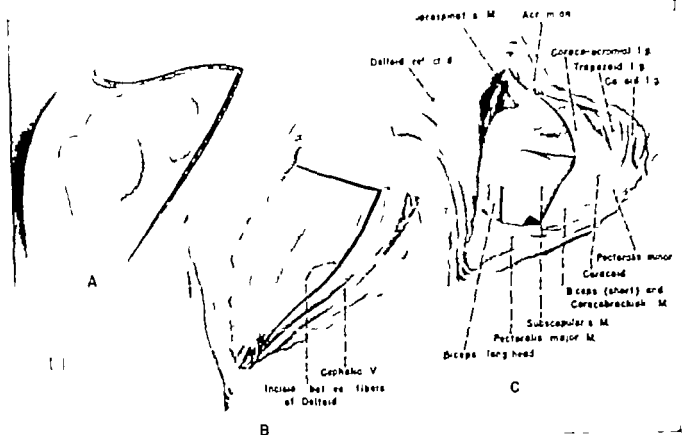


FIG 33: Anterosuperomedial approach of Thompson and Henry (A) Skin incision. If further exposure is desired the incision is continued around the acromion as in the incision of Cubbins et al (B) Line of reflection of split deltoid muscle from the clavicle by sharp dissection indicated (C) Exposure of structures in the anterior, superior and medial aspects of the shoulder region

zontal limb starts over the anterior surface of the acromioclavicular joint proceeds medially along the anterior surface of the outer third of the clavicle and joins the vertical limb at the deltopectoral sulcus just to the inner side of the coracoid process

Separation of the vertical limb's skin edges reveals the cephalic vein lying in the deltopectoral interval. This vein together with a few anterior fibers of the deltoid and the pectoralis major muscle is retracted medially while the deltoid is retracted laterally. Injury to the cephalic vein and the thoraco-acromial artery which lies in the upper end of the incision must be avoided while developing the vertical arm of the incision. The clavicular head of the deltoid is separated from the outer third of the clavicle with a thin sharp osteotome. The entire deltoid mass is then reflected downward and laterally giving an excellent view

of the anterior and the lateral portions of the shoulder region (Fig 337)

Further exposure can be attained by severing the coraco-acromial ligament close to its attachment to the acromion dividing the tip of the coracoid process proximal to the insertion of the coracohumeral ligament, and displacing it downward and medially with its muscle attachments. This step permits access to the anterior surface of the subscapularis muscle and tendon. The anterior and the inferior portions of the fibrous capsule come into view if the tendon is divided an inch medial to its insertion into the lesser tuberosity and then displaced medially. Longitudinal division of the capsule close to the glenoid rim permits exploration of the interior of the glenohumeral joint.

Anterosuperoposterior Approach (Incision of Cubbins et al) If greater ex-

position behind that of the latissimus dorsi. The bilaminar tendon of the pectoralis major muscle can be seen inserting into the crest of the greater tuberosity. Upon opening the roof of the subacromial bursa, its floor, which is adherent to the musculotendinous cuff is exposed (Fig 335).

Short Anterior Approach through the Deltoid Muscle. This approach to the subacromial region is most commonly employed for such minor surgical procedures as removal of calcareous deposits in the musculotendinous cuff, excision of the subacromial bursa, removal of osteophytes on the greater tuberosity, and exploration. The disadvantages of this approach are many. It provides very poor exposure except for the structures immediately beneath the roof of the subacromial bursa; the axillary nerve is very vulnerable to injury; and finally it fails to provide enough room for reparative procedures on the musculotendinous cuff except for the most simple and very small tears.

The sitting position is preferred for this approach. The arm should be draped separately to facilitate free movements of the limb in any desired position. Local anesthesia or Pentothal Sodium intravenously are the anesthetic agents of choice.

Beginning at the acromioclavicular joint, the skin incision is extended vertically downward for a distance not exceeding 2 inches. The muscle fibers are split from above downward in line with the skin incision, exposing the roof of the subacromial bursa. Division of the bursal roof brings into view its floor and the outer surface of the musculotendinous cuff. By external and internal rotation of the arm, the entire insertion of the cuff can be visualized as well as the intertubercular sulcus, the tendon of the long head of the biceps brachii muscle, and the greater and the lesser tuberosities (Fig 336).

A lateral incision similarly gives access to the lateral and the superior aspects of the subacromial region. Lateral incisions are necessary occasionally to reach calcareous

deposits situated far posteriorly. Exposure of the short muscle splitting incision can be increased by stripping subperiosteally an inch of the deltoid muscle on either side of the incision from its bony origin.

The author has supplanted the short anterior vertical skin incision by a 2½ inch horizontal skin incision, placed an inch below the inferior border of the acromioclavicular joint. The proximal 2 inches of the deltoid muscle are exposed by retracting the upper and lower margins. As in the vertical incision, the deltoid muscle is split longitudinally in the line of its fibers for a distance of from 1½ to 2 inches. This skin incision gives a better cosmetic result than the vertical incision. Healing takes place with minimum scarring and separation of skin margins.

Anterosuperomedial Approach (Incision of Thompson and Henry) This is a combination of Roberts' approach to the acromioclavicular joint and the deltopectoral incision. It provides excellent exposure of all the structures in the anterior and the superomedial aspects of the shoulder region. This route practically eliminates danger to the axillary nerve. Ample working room and good accessibility to the important structures are provided. Lesions of the musculotendinous cuff and of the biceps tendon and fractures and fracture-dislocations of the head of the humerus can be cared for readily through this incision. However, extensive reparative work on the musculotendinous cuff is performed best through the superior approaches. The sitting position is preferable with the shoulder well over the side of the operating table and the arm draped separately. Intratracheal anesthesia or Pentothal Sodium intravenously are superior to inhalation and local anesthetics.

The vertical limb of the skin incision is similar to that of the deltopectoral approach. It extends along the anterior border of the deltoid muscle beginning at the inferior margin of the clavicle at the junction of its outer and middle thirds and ending proximal to the deltoid tuberosity. Its hori-

the anterior border of the deltoid does not decrease the efficiency of the muscle. This approach is particularly indicated in reconstruction procedures of the musculotendinous cuff. McLaughlin points out that it facilitates removal of displaced fractured fragments of the humeral head from above rather than pulling them through the brachial plexus from below. Reattachment of the labrum or capsule to the glenoid rim for recurrent dislocations of the shoulder is performed readily from within the scapulohumeral joint. The Nicola procedure also can be performed with greater accuracy because the tunnel for the biceps tendon is made from above downward and the point of exit of the tendon from the head of the humerus is chosen under vision. Finally, this route enables better restoration of fractured fragments to their normal anatomic position.

The position of choice is with the patient seated and the shoulder extending well beyond the edge of the operating table. A sandbag is placed under the shoulder, and the table is tilted away from the operator. The arm is draped separately to facilitate movement of the limb in any desired position. Intratracheal anesthesia is preferred.

Beginning at the posterior border of the acromion and lateral to the acromioclavicular joint the skin incision is carried across the top of the shoulder to a point 3 to 5 centimeters in front of the anterior border of the acromion. The incision is developed anteriorly through the deltoid muscle exposing the roof of the subacromial bursa and the coraco-acromial ligament. Incision of the superior bursal wall brings into view the greater portion of the outer surface of the musculotendinous cuff only the most posterior and inferior portions remaining obscured.

Next the superior limb of the incision is deepened to the bone and the acromion is divided with a thin blade osteotome. For simple repair of the musculotendinous cuff, an oblique osteotomy is done beginning midway between the acromioclavicular joint

and the lateral border of the acromion and directed laterally to emerge at the lateral tip of the acromion. In cases requiring more extensive surgery and exploration of the interior of the glenohumeral joint the osteotomy is directed anteroposteriorly by reflecting the deltoid and the outer fragment of the acromion laterally. All the superior and lateral aspects of the shoulder region are visualized as far as the surgical neck.

Access to the interior of the joint is achieved by a longitudinal incision in the musculotendinous cuff in the interval between the subscapularis and the supraspinatus muscles and parallel with the fibers of the coracohumeral ligament. There is no need to replace the detached fragment of the acromion process; it is more advantageous to dissect it free and discard it. By reducing the size of the overhanging acromion, impingement of the greater tuberosity or the repaired cuff against the acromion is less likely to occur upon elevation of the extremity. McLaughlin emphasizes that a small acromion means a less prominent fulcrum, over which the humeral head may be levered out of the glenoid cavity. Hence, the tendency for recurrences is minimized in operative procedures for recurrent dislocations. Moreover the postoperative course is less painful, and rehabilitation of the patient is quicker when the detached fragment is removed.

Saber-cut Incision (Codman) This incision, first described by Codman, provides an excellent exposure of the subacromial bursa and the whole musculotendinous cuff. It also brings into view the extra-articular portion of the tendon of the long head of the biceps brachii muscle. All regions of the inside of the joint can be explored by opening the capsule of the joint in the same manner as described in the transacromial approach. However, it is a mutilating incision and possesses no advantages over the transacromial route.

Beginning from 3 to 4 centimeters below the spine of the scapula the skin incision proceeds anteriorly over the top of the

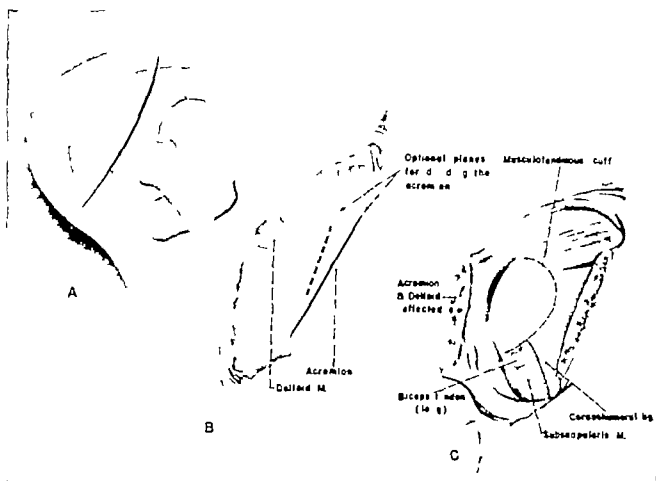


FIG 338 Transacromial incision (McLaughlin) (A) Skin incision (B) Planes through which acromion may be divided to gain access to the subacromial region (C) Structures exposed after the deltoid is reflected downward and outward.

posure of the lateral or exposure of the posterior aspect of the shoulder region is desired the horizontal arm of the antero-superomedial incision is projected around the acromion process and along the lateral half of the spine of the scapula (Fig 337-A). By sharp dissection with a thin osteotome the origin of the deltoid is stripped from the anterior border of the clavicle the acromion process and the spine of the scapula. Downward reflection of the entire deltoid permits complete visualization of the anterior lateral and posterior shoulder regions. By dividing longitudinally the posterior portion of the musculotendinous cuff proximal to its insertion into the greater tuberosity the interior of the glenohumeral joint can be inspected.

The last two approaches are particularly useful for fractures, fracture-dislocations, arthrodesis of the glenohumeral joint, repair

of tears of the musculotendinous cuff, recurrent dislocation of the scapulohumeral joint lesions of the long head of the biceps brachii muscle and tumors of the upper end of the humerus. Giant-cell tumors and cysts of upper end of the humerus are made readily accessible by these routes.

SUPERIOR APPROACHES

Transacromial Approach (McLaughlin) This is the most commonly used superior approach providing adequate exposure of the entire superior shoulder region. Since the skin incision parallels the skin creases there is little spreading of the scar hence the cosmetic result is good. Injury to the axillary nerve is not likely to occur if all muscle splitting is done from above downward starting at the anterior margin of the acromion.

Moreover separation of muscle fibers near

(the superior approaches) with the arm draped separately and the operating table tilted away from the surgeon. Intratracheal anesthesia is preferred.

Beginning at the acromioclavicular joint the skin incision proceeds posteriorly over

by blunt dissection. The deltoid is divided 1 centimeter from its scapular origin and detached subperiosteally as far as the acromioclavicular joint. By retracting the detached portion of the muscle laterally the posterior aspect of the joint is brought into

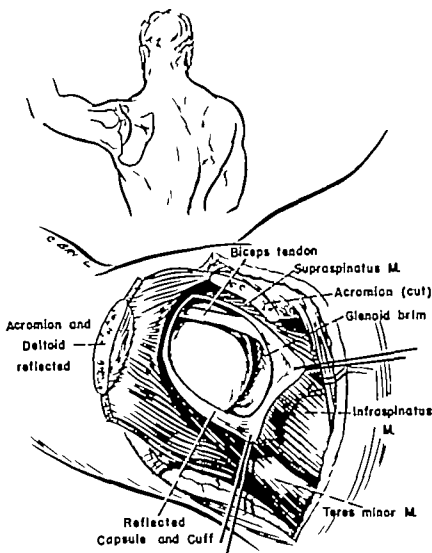


FIG. 339 Kocher's posterior route to the scapulohumeral joint. Mobilization of the posterior portion of the deltoid muscle may be achieved either by subperiosteal detachment from the spine and osteotomy of the outer end of the acromion or by continuing the subperiosteal detachment from the spine around the acromion as far as the acromioclavicular joint.

the top of the acromion to the spine of the scapula then gently curves downward and outward to a point about 4 centimeters above the posterior fold of the axilla. Next the posterior border of the deltoid muscle is identified and the interval between the deltoid and the deeper muscles is developed

view. Care must be exercised at this point to avoid injury to the axillary nerve and its branches and to the posterior axillary artery as they emerge from the quadrilateral space which can now be observed readily.

Some surgeons prefer to divide the lateral half of the acromion and to displace it

shoulder in line with the acromioclavicular joint and ends roughly from 3 to 4 centimeters below the border of the acromion process. Anteriorly and posteriorly the incision is deepened through the fibers of the deltoid muscle. At this point care must be exercised to avoid damage to the axillary nerve in front and behind.

The acromioclavicular ligaments are severed and the acromion is divided in a line directed anteroposteriorly with a thin osteotome. The detached acromion, together with the deltoid muscle, is retracted downward, bringing into view the subacromial bursa, the musculotendinous cuff, the intertubercular sulcus and the long head of the biceps tendon.

The topographic anatomy of the supra scapular nerve and artery must be kept in mind while displacing the deltoid mass downward. Forceful retraction may inflict severe injury to these structures as they traverse the suprascapular notch. On closure of the incision the detached acromion process is sutured or wired to its normal anatomic position and the divided edges of the superior acromioclavicular ligament are approximated by interrupted sutures.

Superior Approach of Henry. This route provides excellent exposure of the superior, anterior, lateral and posterior aspects of the shoulder region without severing the acromion process. Minor cuff tears can be repaired readily; the lesions of the tendon of the long head of the biceps brachii can be dealt with; fracture fragments reassembled; fracture-dislocations reduced; and operations for recurrent dislocation of the shoulder performed. However, as previously stated, the transacromial approach gives more adequate exposure for all the afore-mentioned surgical procedures and is particularly desirable in repairing extensive tears of the musculotendinous cuff. The position of the patient and preferred anesthesia are similar to those of other superior approaches.

Essentially the skin incision is a continuation of the deltopectoral incision over the top of the shoulder to a point just below

the spine of the scapula. As in the deltopectoral approach, the interval between the deltoid and the pectoralis major muscle is developed to the lower margin of the clavicle. The cephalic vein with 1 centimeter of the anterior border of the deltoid, together with the pectoralis major, is retracted medially and the deltoid muscle is retracted laterally. By sharp dissection with a thin osteotome, the origin of the deltoid is stripped from the anterior border of the outer one-third of the clavicle and the lateral edge of the acromion. The deltoid muscle is then retracted downward and outward. If more exposure is desired, the origin of the deltoid may be detached from the entire acromion and lateral one-third of the spine of the scapula.

POSTERIOR APPROACHES

As noted above, the posterior aspect of the shoulder region may be visualized through the superior routes or by extension of the anterosuperomedial incisions around the lateral and the posterior margins of the acromion and the lateral portion of the spine of the scapula (Cubbin's incision). However, it is sometimes desirable to limit exposure to the posterior aspect of the shoulder, making superior approaches and Cubbin's incision not justified.

The most commonly used posterior route is the one of Kocher. Several others have been described (the approaches of Bennett, Harmon and of Rowe and Yee) but they possess no particular features which would make them superior to the Kocher approach. Yee and Rowe emphasize the value of their approach to the postero-inferior aspect of the glenohumeral joint along anatomic planes with little risk of injury to the axillary veins and branches supplying the deltoid and the teres minor muscles.

Posterior Approach of Kocher. The patient may be placed face down with a sandbag under the shoulder, the head turned to the opposite side, and the outstretched arm supported on an arm board or in the sitting position (as described for

the superior approaches) with the arm draped separately and the operating table tilted away from the surgeon. Intratracheal anesthesia is preferred.

Beginning at the acromioclavicular joint the skin incision proceeds posteriorly over

by blunt dissection. The deltoid is divided 1 centimeter from its capsular origin and detached subperiosteally as far as the acromioclavicular joint. By retracting the detached portion of the muscle laterally the posterior aspect of the joint is brought into

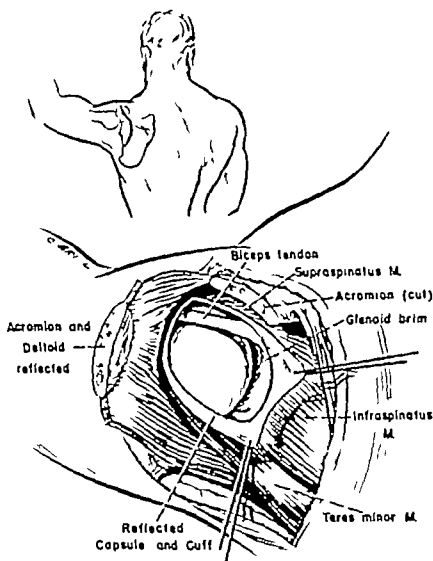


FIG. 339 Kocher's posterior route to the scapulohumeral joint. Mobilization of the posterior portion of the deltoid muscle may be achieved either by subperiosteal detachment from the spine and osteotomy of the outer end of the acromion or by continuing the subperiosteal detachment from the spine around the acromion as far as the acromioclavicular joint.

the top of the acromion to the spine of the scapula then gently curves downward and outward to a point about 4 centimeters above the posterior fold of the axilla. Next the posterior border of the deltoid muscle is identified and the interval between the deltoid and the deeper muscles is developed

view. Care must be exercised at this point to avoid injury to the axillary nerve and its branches and to the posterior axillary artery as they emerge from the quadrilateral space which can now be observed readily.

Some surgeons prefer to divide the lateral half of the acromion and to displace it

laterally with the attached deltoid instead of stripping the deltoid as far as the acromioclavicular joint. If a view of the posterior aspect of the interior of the glenohumeral joint is desired, the tendons of the infraspinatus, the teres minor and a portion of the tendon of the supraspinatus muscles may be divided longitudinally, together with the capsule close to their insertion into the greater tuberosity leaving sufficient tissue for easy reattachment of the severed ends. The intracapsular portion of the tendon of the long head of the biceps brachii muscle must not be cut in this last step of the procedure (Fig. 339).

Posterior Approach of Rowe and Yee
The patient is placed in the prone position with both arms abducted and supported on arm boards. A large sandbag or small pillow is placed under the shoulder to be exposed and the patient's head is turned to the opposite side. Intratracheal anesthesia is far superior to any of the inhalation anesthetics.

Beginning at the junction of the middle and the inner thirds of the spine of the scapula, the skin incision is continued along the spine. The incision then curves gently downward over the posterior aspect of the shoulder joint for approximately 4 inches. By retracting the skin medially, the scapular origin of the deltoid is visualized. This is then stripped subperiosteally from the spine of the scapula. The deltoid is split downward for approximately 3 inches about $1\frac{1}{2}$ inches from its medial border. The lateral triangular muscle flap thus formed is retracted laterally exposing the infraspinatus and the teres minor muscles. The interval between these two muscles is developed by blunt dissection and the infraspinatus is similarly separated from the underlying fibrous capsule. The tendon of the infraspinatus muscle is divided $\frac{1}{2}$ inch from its insertion into the great tuberosity and retracted medially. Retraction of the teres minor downward now brings into view the entire posterior and inferior portions of the capsule. Care must be exercised to limit the vertical incision severing the infra-

spinatus tendon to the upper border of the teres minor muscle. Should it extend beyond this point the axillary nerve traversing the quadrilateral space may be injured (Fig. 340).

A vertical incision in the capsule permits adequate exploration of the inside of the posterior and the inferior aspects of the joint. Through this route Rowe and Yee reattached the torn labrum to the posterior lip of the glenoid brim in two cases of recurrent posterior dislocation of the shoulder. In the same manner that Bankart reattaches the anterior labrum to the anterior glenoid brim for recurrent anterior dislocations.

APPROACH TO THE STERNOCLAVICULAR JOINT

The skin incision begins over the clavicular head of the sternocleidomastoid muscle, just above the upper border of the clavicle and extends medially to the outer border of the sternal head. It then curves gently downward on the anterior surface of the sternum for $1\frac{1}{2}$ inches. Retraction of the skin margins exposes the tendinous insertions of both heads of the sternocleidomastoid muscle. By sharp subperiosteal dissection the sternocleidomastoid and the pectoralis major muscles are reflected from the clavicle upward and downward respectively thereby exposing the fibrous capsule of the sternoclavicular joint. A horizontal or curved incision is made in the articular capsule exposing adequately the interior of the joint.

APPROACH TO THE ACROMIOCLAVICULAR JOINT (INFRACLAVICULAR ROUTE OF CUBBINS ET AL.)

The skin incision begins at the angle of the acromion, proceeds medially along the lower border of the acromion, the acromioclavicular joint and the outer third of the clavicle to the deltopectoral cleft where it swings downward and outward for a distance of from 1 to 2 inches following the anterior border of the deltoid. Next the interval between the deltoid and the pec-

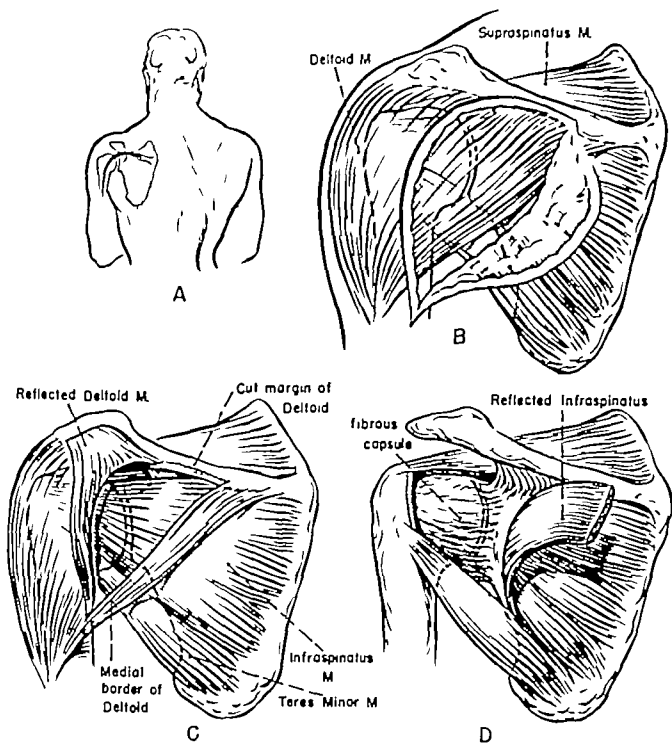


FIG. 340 Posterior approach (Rowe and Yee) (A) Skin incision. (B) Medial reflection of skin exposing posterior portion of deltoid (C) Deltoid split longitudinally 1 and $\frac{1}{2}$ inches from its medial border and detached subperiosteally from the spine then retracted laterally (D) Infraspinatus tendon is severed $\frac{1}{2}$ inch from its insertion into the greater tuberosity and retracted medially exposing the posterior portion of the fibrous capsule

toralis major muscles is developed to the clavicle

At this point care must be taken not to injure the cephalic vein. The origin of the deltoid is stripped by sharp subperiosteal dissection from the anterior margin of the

clavicle and the acromion and reflected downward. In a similar manner the trapezius is stripped from the superior surface of the clavicle and the acromion and retracted upward. The pectoralis major, together with the cephalic vein and 1 centi

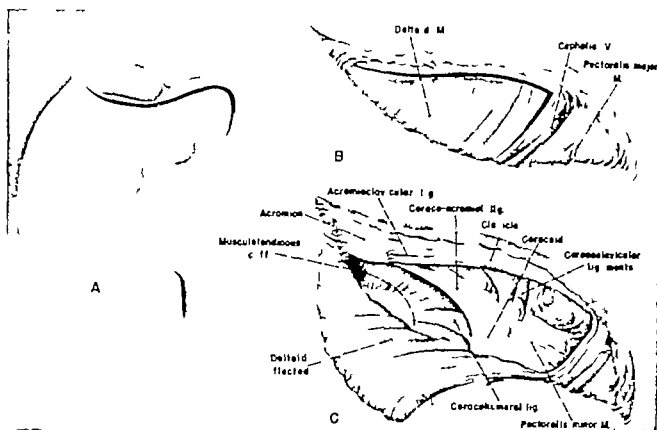


FIG 341 Intraclavicular approach to the acromioclavicular joint (Cubbins et al.) (A) Skin incision (B) Line of subperiosteal stripping of the origin of deltoid muscle from the clavicle (C) Exposure of the acromioclavicular joint and coracoclavicular ligaments

meter of the anterior border of the deltoid is retracted medially and downward. Hence the coracoid process, the coraco-acromial ligament, the coracoclavicular ligaments (trapezoid and conoid ligaments) and the articular capsule of the acromioclavicular joint are exposed (Fig. 341).

INCISION TO EXPOSE THE CLAVICLE

The patient lies supine with a small sand bag or a folded sheet between the shoulder blades. The head is turned to the opposite side.

The clavicle lies for its entire length in a very superficial position. It can be palpated readily beneath the skin from its acromial to its sternal end. Exposure of this bone is achieved best by an incision in the supra-clavicular fossa running along the antero-superior border of the clavicle from the sternal to the acromial end. The skin incision is carried through the platysma muscle and the periosteum down to the bone. By sharp dissection with a thin blade osteo-

tome the periosteum and attached clavicular head of the sternocleidomastoid muscle and the trapezius muscle are stripped from the shaft and retracted proximally in a like manner the pectoralis major and the deltoid muscles are stripped from the clavicle and reflected distally.

INCISIONS TO EXPOSE THE SCAPULA

Occasionally it becomes necessary to expose part of or all the scapula. Benign lesions such as osteochondromas which require excision are encountered occasionally on the ventral or dorsal surface of the scapula.

Scapulectomy may be indicated for some malignant bone lesions. Profuse bleeding can be prevented by sharp subperiosteal stripping of the overlying muscles from either surface of the scapula.

EXPOSURE OF VERTEBRAL BORDER OF SCAPULA

The patient is prone with a flat sandbag under the shoulder on the side of the opera-

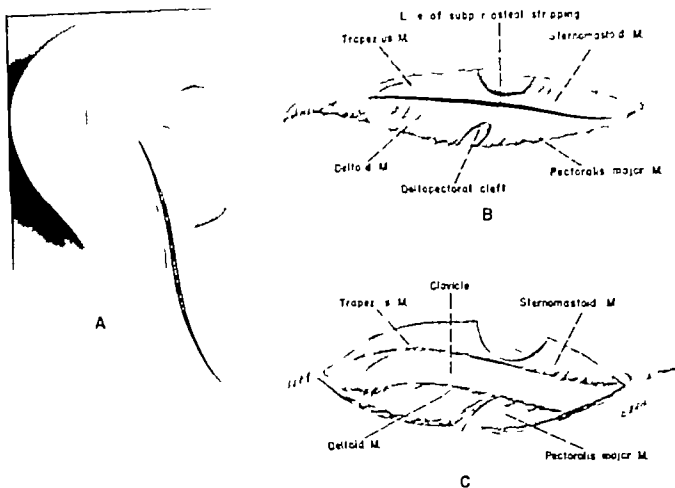


FIG. 342 Exposure of entire clavicle

tion and the arm is draped separately to the side. Intratracheal anesthesia facilitates the operative procedure.

The skin incision begins at the superior angle of the scapula, continues distally along its vertebral border and ends at the inferior angle. Next the periosteum is incised horizontally over the root of the spine of the scapula, beginning at its vertebral margin for a distance of 2 inches. The periosteum and the attached trapezius are stripped from the spine with a sharp osteotome. The incision is continued upward and medially through the substance of the trapezius muscle in the line of its fibers for 1 inch. A triangular flap of muscle is thus formed which when reflected upward and medially exposes the muscles inserting into the vertebral border of the scapula.

The above mentioned muscles the levator scapulae, the rhomboid minor, the rhomboid major, the supraspinatus, the infraspinatus, the teres major and the teres

minor are incised to the bone close to the vertebral border and then detached subperiosteally with a sharp osteotome exposing the vertebral margin of the scapula. Subperiosteal stripping and reflection of the muscles may be extended on the dorsal surface of the scapula as far laterally as desired. If the ventral surface of the scapula must be exposed the subperiosteal stripping is carried around the vertebral border to the ventral aspect and the muscles attached to the undersurface of the scapula are stripped away (Fig. 343).

Kocher's Incision. Exposure of the entire scapula may be achieved by Kocher's incision. The vertical limb of the incision begins at the superior angle, continues along the vertebral border and ends at the inferior angle of the scapula. The horizontal limb runs along the superior surface of the spine of the scapula beginning at the vertebral border and ending at the lateral margin of the acromion. The horizontal incision is

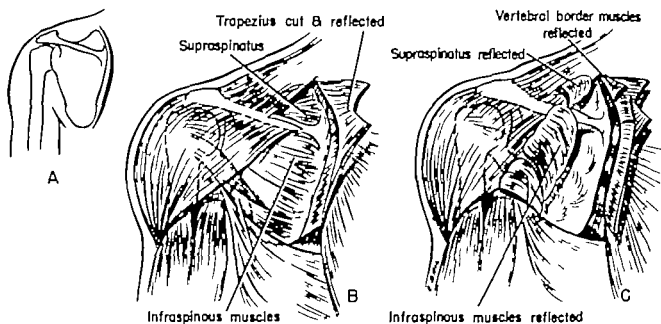


FIG 343 Exposure of the vertebral border of scapula.

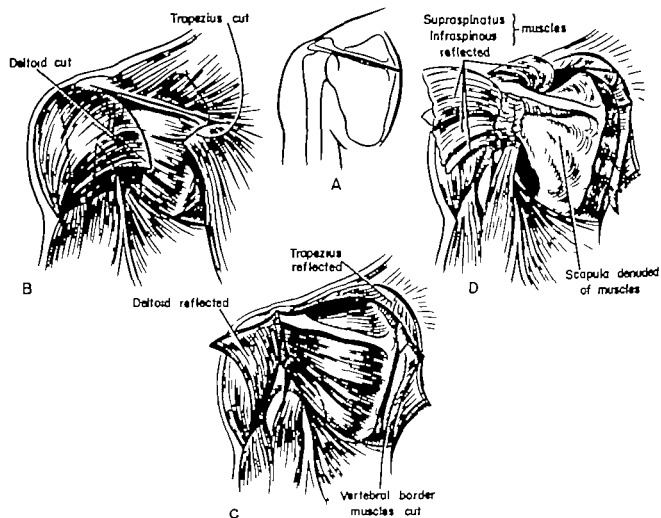


FIG 344 Exposure of entire scapula (Kocher)

deepened through the periosteum of the spine to the bone and continues medially through the substance of the trapezius muscle for 2 to 3 centimeters.

The periosteum and the attached trapezius muscle are detached from the spine and reflected upward by sharp subperiosteal dissection. In a similar manner, the deltoid is detached and reflected downward and laterally. All muscles are divided close to their line of attachment to the entire vertebral border and stripped away subperiosteally. By reflecting the muscle masses on the ventral and dorsal surfaces the whole of the scapula may be exposed (Fig. 344).

ARTHRODESIS OF THE SCAPULOHUMERAL JOINT

GENERAL CONSIDERATIONS

There are few indications for arthrodesis of the scapulohumeral joint. However, when justified and properly performed the procedure gives gratifying results.

It is performed for the following conditions:

- 1 Paralysis of the deltoid muscle resulting from anterior poliomyelitis
- 2 Paralysis of the deltoid secondary to peripheral nerve (axillary nerve) or brachial plexus injury
- 3 Traumatic lesions of the upper end of the humerus
- 4 Complete or massive avulsion of the musculotendinous cuff in which some type of repair is not possible
- 5 Tuberculosis of the scapulohumeral joint
- 6 Neurotrophic disorders (syphilis)
- 7 Infectious arthritis associated with destruction of the joint and pain

Paralysis of the deltoid muscle and tuberculosis of the glenohumeral joint are the most common of the afore-mentioned indications. One is rarely justified in fusing the shoulder joint for traumatic disorders. As has been pointed out (Chap. 8) malalignment of fractured fragments except in extreme cases, does not preclude good pain

less shoulder function. In fracture-dislocation with complete separation of the humeral head from the shaft, the head may be removed and the cuff may be attached to the rounded end of the shaft. Such procedure gives a result superior to fusion of the shoulder joint. Moreover, such lesions usually occur in the late decades of life, when an arthrodesing procedure and the subsequent rehabilitation of the extremity may prove to be too formidable.

Some workers prescribe arthrodesis in cases of massive or complete avulsion of the musculotendinous cuff, believing that reattachment of the cuff results in a less useful extremity. However, this has not been the experience of the author who agrees with McLaughlin that reattachment of the cuff to some point on the head of the humerus can be achieved in most of these cases and that the results attained are more desirable than those of joint fusion (see Chap. 4).

Arthrodesis is the treatment of choice for tuberculosis of the scapulohumeral joint. Only complete obliteration of the joint can assure eradication of the disease. However, it never should be performed during the active stages. A course of streptomycin before and after the procedure minimizes the possibility of secondary infection and sinus formation and favors the healing of affected tissues. Arthrodesis should be withheld in children until sufficient growth of the humeral head and the scapula has occurred. The operation is rarely justifiable before the age of 6 years.

Adequate evaluation of the musculature of the entire upper extremity is imperative before fusion in cases with paralysis of the deltoid muscle. Good power in the serratus anticus and the trapezius muscles is a prime requisite if arthrodesis is contemplated, since these muscles will be responsible for motivating the shoulder girdle following the fusion. Moreover, it must be pointed out again that the chief function of the shoulder girdle is to place the hand in positions for function where it can perform required

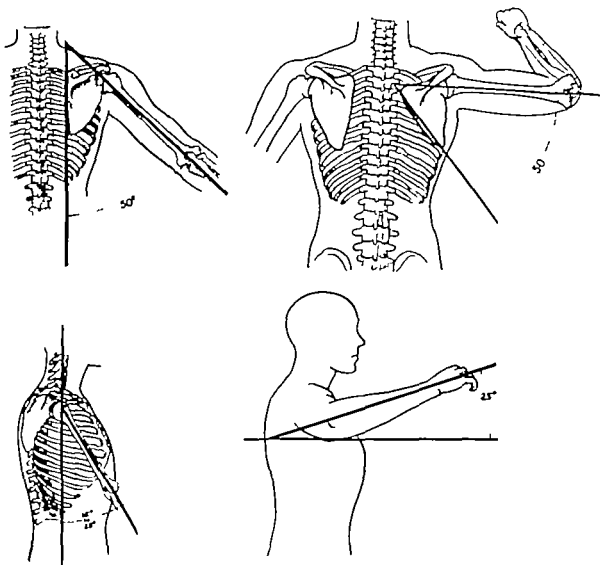


FIG 345 (*Top left*) The recommended position of abduction with the scapula in normal anatomic position the humerus is abducted 50 degrees from the vertebral border of the scapula (viewed from behind) (*Top right*) Recommended position of immobilization of the arm after arthrodesis. Observe the relation of the humerus to the scapula and to the trunk. (*Bottom left*) Recommended amount of flexion with scapula in normal anatomic position. (*Bottom right*) Side view exhibiting recommended positions of flexion and rotation (Research Committee of the American Orthopedic Association J Bone & Joint Surg 24 704 705 and 706)

duties. If paralysis of the hand exists arthrodesis of the scapulohumeral joint (to permit the patient to place the hand in various positions of function through the medium of scapular motion) is a needless operation unless done to provide a sense of stability.

It must be appreciated that arthrodesis of the glenohumeral joint is not a simple procedure and that the results are often far from satisfactory. Several factors are responsible. The architectural structure of the

bony joint components does not lend itself readily to surgical fusion. The glenoid cavity is small. It provides only a meager surface of cancellous bone when denuded of its cartilage and only a small portion of the large spherical head is in direct contact with the glenoid cavity. The overhanging acromion is at a considerable distance from the head of the humerus when the latter is in its normal anatomic relation to the glenoid fossa. Lack of knowledge or dis-

regard of the correct position of the humerus in relation to the vertebral border of the scapula (necessary for optimum function) and such inadequate postoperative management as improperly applied casts and short periods of immobilization are also responsible for unsatisfactory shoulder fusions.

In 1942 the Research Committee of the American Orthopedic Association conducted a study on 145 cases of infantile paralysis. Of this group an arthrodesis of one shoulder had been performed on 100. A bilateral arthrodesis in 1 case, some type of muscle transplantation in 43, stabilization of a subluxated shoulder by the Nicola operation for 2, and stabilization of the shoulders by tying the scapulae together with a fascial sling for 1.

This investigation permitted formulation of the following pertinent conclusions: the most common causes leading to poor function were poor positioning of the humerus in relation to the scapula postoperatively and disregard of the position of rotation of the humerus.

Younger individuals developed better scapular movements than older individuals. Scapular winging did not occur with the shoulder fused in 45° abduction in the presence of good power in the muscles controlling the scapula (upper trapezius and serratus anterior muscles). Abduction was possible to 90° and the arm came readily to the side. Slightly greater abduction improved the functional result but not the cosmetic effect. In view of these findings boys under 12 years of age should be fused in 55° abduction. This results in slight winging of the scapula but gives a more useful range of scapular motion. Girls should be fused in 45° abduction because this position gives the optimum cosmetic result.

Optimum function depends upon good power in the upper portion of the trapezius and the upper two-thirds of the serratus anterior muscles. The upper fibers of the trapezius muscle raise the acromion process and the entire shoulder girdle pivots on the sternoclavicular joint. The trapezius

muscle is able to perform the above function even in the absence of power in the serratus anterior but it can abduct the arm only 45°. It was also noted that when the humerus was fused at 45° or over from the vertebral border of the scapula in the absence of power in the serratus anterior the weight of the arm may rotate and depress the outer portion of the scapula thereby stretching and reducing the efficiency of the trapezius. It becomes apparent that with a paralyzed serratus anterior the optimum position of abduction of the humerus from the scapula is 30°.

Fusion of the shoulder is justified with only fair power in the trapezius and paralysis of all other muscles because some functional improvement is always achieved. Also a flail shoulder usually rides at a higher level than the normal shoulder and arthrodesis restores the normal alignment thereby improving the cosmetic effect.

Adduction contractures of the soft tissues about the scapulohumeral joint (resulting from the extremity's being in an extreme adducted position close to the chest wall for long intervals) may preclude positioning of the humerus in the desired degrees of abduction unless the tissues are first released or stretched.

True abduction at operation is determined by the angle that the humerus makes with the vertebral border of the scapula. Having attained the true abduction angle desired the scapula and the humerus can be elevated further as a single unit so that the arm is abducted 70° to 90° from the trunk. This position favors relaxation of the trapezius and the serratus anterior muscles. However due to the tendency of the scapula to rotate within the plaster cast this relationship may be difficult to maintain or even lost. Internal fixation between the head of the humerus and the neck of the scapula or between the acromion process and the head is advocated to ensure maintenance of the desired abduction angle.

Care should be exercised to avoid excessive flexion of the humerus in relation to the

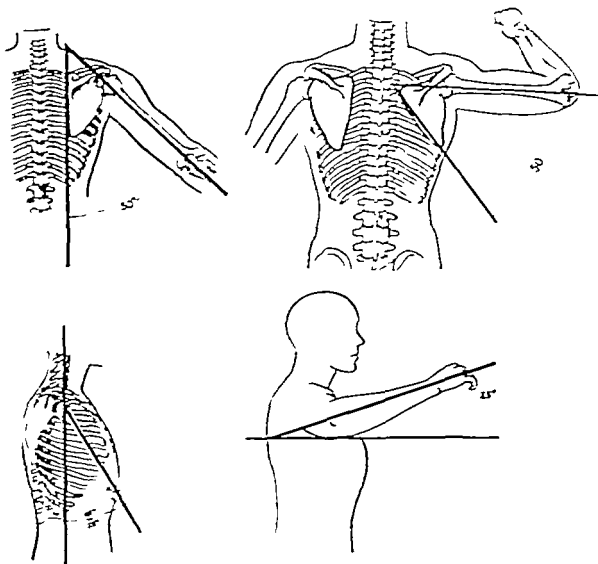


FIG. 34 (Top left) The recommended position of abduction with the scapula in normal anatomic position, the humerus is abducted 40 degrees from the vertebral border of the scapula (viewed from behind). (Top right) Recommended position of immobilization of the arm after arthrodesis. Observe the relation of the humerus to the scapula and to the trunk. (Bottom left) Recommended amount of flexion with scapula in normal anatomic position. (Bottom right) Side view exhibiting recommended positions of flexion and rotation. (Research Committee of the American Orthopedic Association J. Bone & Joint Surg. 24: 34 '05 and '06)

dules. If paralysis of the hand exists arthrodesis of the scapulohumeral joint (to permit the patient to place the hand in various positions of function through the medium of scapular motion) is a needless operation unless done to provide a sense of stability.

It must be appreciated that arthrodesis of the glenohumeral joint is not a simple procedure and that the results are often far from satisfactory. Several factors are responsible. The architectural structure of the

bone joint components does not lend itself readily to surgical fusion; the glenoid cavity is small; it provides only a meager surface of cancellous bone when denuded of its cartilage; and only a small portion of the large spherical head is in direct contact with the glenoid cavity. The overhanging acromion is at a considerable distance from the head of the humerus when the latter is in its normal anatomic relation to the glenoid fossa. Lack of knowledge or dis-

regard of the correct position of the humerus in relation to the vertebral border of the scapula (necessary for optimum function) and such inadequate postoperative management as improperly applied casts and short periods of immobilization are all responsible for unsatisfactory shoulder fusions.

In 1942 the Research Committee of the American Orthopedic Association conducted a study on 148 cases of infantile paralysis. Of this group an arthrodesis of one shoulder had been performed on 100, a bilateral arthrodesis in 1 case, some type of muscle transplantation in 43, stabilization of a luxated shoulder by the Nicoli operation for 2, and stabilization of the shoulders by tying the scapulae together with a fascial sling for 1.

This investigation permitted formulation of the following pertinent conclusions: the most common causes leading to poor function were poor positioning of the humerus in relation to the scapula postoperatively and disregard of the position of rotation of the humerus.

Younger individuals developed better scapular movements than older individuals. Scapular winging did not occur with the shoulder fused in 45° abduction in the presence of good power in the muscles controlling the scapula (upper trapezius and serratus anterior muscles); abduction was possible to 90° and the arm came readily to the side. Slightly greater abduction improved the functional result but not the cosmetic effect. In view of these findings boys under 12 years of age should be fused in 55° abduction. This results in slight winging of the scapula but gives a more useful range of scapular motion. Girls should be fused in 45° abduction because this position gives the optimum cosmetic result.

Optimum function depends upon good power in the upper portion of the trapezius and the upper two-thirds of the serratus anterior muscles. The upper fibers of the trapezius muscle raise the acromion process and the entire shoulder girdle pivots on the sternoclavicular joint. The trapezius

muscle is able to perform the above function even in the absence of power in the serratus anterior but it can abduct the arm only 45°. It was also noted that when the humerus was fused at 45° or over from the vertebral border of the scapula in the absence of power in the serratus anterior the weight of the arm may rotate and depress the outer portion of the scapula thereby stretching and reducing the efficiency of the trapezius. It becomes apparent that with a paralyzed serratus anterior the optimum position of abduction of the humerus from the scapula is 30°.

Fusion of the shoulder is justified with only fair power in the trapezius and paralysis of all other muscles because some functional improvement is always achieved. Also a fused shoulder usually rides at a higher level than the normal shoulder and arthrodesis restores the normal alignment thereby improving the cosmetic effect.

Adduction contractures of the soft tissues about the scapulohumeral joint (resulting from the extremity's being in an extreme adducted position close to the chest wall for long intervals) may preclude positioning of the humerus in the desired degrees of abduction unless the tissues are first released or stretched.

True abduction at operation is determined by the angle that the humerus makes with the vertebral border of the scapula. Having attained the true abduction angle desired the scapula and the humerus can be elevated further as a single unit so that the arm is abducted 70° to 90° from the trunk. This position favors relaxation of the trapezius and the serratus anterior muscles. However due to the tendency of the scapula to rotate within the plaster cast this relationship may be difficult to maintain or even lost. Internal fixation between the head of the humerus and the neck of the scapula or between the acromion process and the head is advocated to ensure maintenance of the desired abduction angle.

Care should be exercised to avoid excessive flexion of the humerus in relation to the

scapula, a position which causes winging of the scapula and stretching of the serratus anterior muscle. The optimum position of flexion in girls is from 10° to 15° such a position permits the arm to be flat against the trunk. In boys, flexion of 25° increases the usefulness of the extremity but sacrifices slightly the cosmetic effect. As a rule, an arm placed in from 70° to 90° abduction and from 35° to 40° flexion following operation results in the desired flexion of the humerus to the vertebral border of the scapula. Some degree of flexion is essential for good performance of the shoulder girdle. Without it, the arm is in a useless position.

Poor function results from excessive internal or external rotation of the humerus in the abducted position. Excessive external rotation is worse than excessive internal rotation. Fifteen degrees internal rotation without abducting the arm beyond 35° or 40° permits the hand to reach the top of the head. For most cases, the optimum position of function is from 45° to 55° abduction of the humerus from the vertebral border of the scapula, from 70° to 90° of abduction of the arm from the trunk, from 15° to 25° forward flexion of the arm from the scapula, and from 25° to 30° upward tilt of the flexed forearm above the horizontal plane (Fig. 345).

If a weak elbow and hand exist on the opposite side, the optimum position of function of the fused shoulder is in greater internal rotation but not exceeding 45° .

No evidence was uncovered which pointed to deleterious effect on scoliosis by shoulder arthrodesis. However scapular movements are restricted by scoliosis. If scoliosis is present with the convexity toward the affected side and the shoulder is fused in too much flexion, excessive winging results and the deformity becomes more obvious.

Function is better in individuals whose shoulders are fused before the age of 12 years. Fusion may be done as early as 6 years of age. Disturbance of growth does not generally occur in young children following arthrodesis. In cases requiring bi-

lateral shoulder fusion, the position should permit the hands to be brought together. The weaker side should be fused in more internal rotation when the muscle power differs in the two shoulders. If the muscle power is the same, in right handed individuals the left is placed in a position of greater internal rotation. In left handed individuals, the right is fused in a position of greater internal rotation.

Arthrodesis is justified in completely flail extremities. The advantages cited were ease of turning in bed, getting into a coat with greater ease, the ability to use the hand to steady a piece of paper while writing, and a sense of stability in the shoulder girdle. The optimum position of function in individuals with flail or weak elbows is only slight abduction of the humerus from the scapula and from 40° to 45° of internal rotation. In many instances arthrodesis of the shoulder increases the flexion power of the elbow.

The desired relationship of the humerus to the scapula may be lost in plaster cast. Improved function occurs in arthrodesis regardless of the method employed when the arm is placed in the correct position of function and adequate postoperative management is provided. The committee recommends that some type of internal fixation be used to maintain the desired position.

Age (under 10 years) is not a significant factor in failure of fusion; rather, short periods of immobilization are more likely to result in nonunion. Three months should be the minimum period of fixation. Immobilization should be continued for 2 more months if bony union is not achieved at the end of this period. Regardless of the presence of a bony fusion or a fibrous ankylosis, the glenohumeral joint is stabilized permanently in a fixed position after five months. Hence plaster or any other type of fixation is not indicated 5 months postoperatively. After bony union is demonstrable roentgenographically no further change occurs in the humeroscapular angle although from 10 to 20 of abduction may be lost in the first

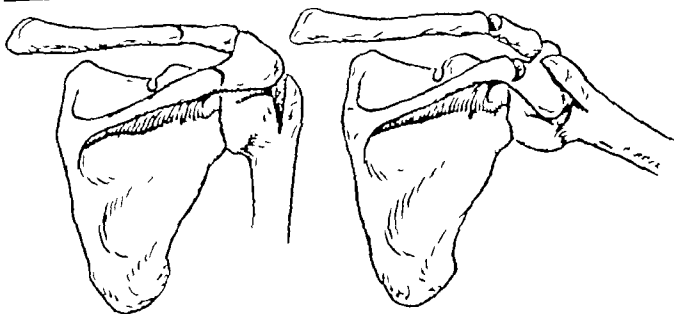


FIG. 346. Extra articular arthrodesis (Watson Jones)

5 months. Fibrous ankylosis is undesirable because pain and fatigue may occur. More over most of these cases disclose only slight abduction a position which permits poor scapular motion.

ARTHRODESIS PROCEDURES

Numerous operations have been described for achieving arthrodesis of the glenohumeral joint. Essentially the operations fall into three categories: extra-articular arthrodesis, intra-articular arthrodesis and a combination of both methods.

Extra-articular arthrodesis is the procedure of choice in tuberculosis of the glenohumeral joint. The combined procedure may be employed in nontuberculous cases. Intra-articular arthrodesis alone results in a high percentage of failures. The author has found that by utilizing the principle of compression, solid bony ankylosis of the joint is obtained in a considerably shorter period than when compression was not employed. Moreover, osseous union occurred in all instances in which compression was added to the procedure.

Extra-articular Arthrodesis. The two most commonly used methods are the Watson Jones and the Putti techniques.

EXTRA-ARTICULAR ARTHRODESIS OF WAT

SON JONES. This procedure is more easily performed with the patient in a sitting position with the shoulder well over the side of the table and the arm draped separately to be manipulated readily in any direction.

A lateral skin incision is made beginning on the top of the shoulder 3 inches above and ending 3 inches below the acromioclavicular joint. The incision is deepened from above downward through the deltoid muscle parallel with its fibers. If care is exercised not to split the deltoid more than 4 centimeters from the lateral margin of the acromion the axillary nerve will not be severed. The origin of the deltoid is then reflected from the acromion, the spine of the scapula and the clavicle by sharp dissection with a thin blade osteotome. Next the acromion on its superior and inferior surfaces and the lateral third of the clavicle are denuded to cancellous bone.

Just distal to the line of insertion of the musculotendinous cuff into the greater tuberosity, a bone flap (measuring 1 inch in width and 2 inches in length) is elevated outward from the anterolateral aspect of the humerus with a wide osteotome. An incomplete osteotomy is made in the outer third of the clavicle and the root of the acromion. The acromion and the distal end of the clavicle

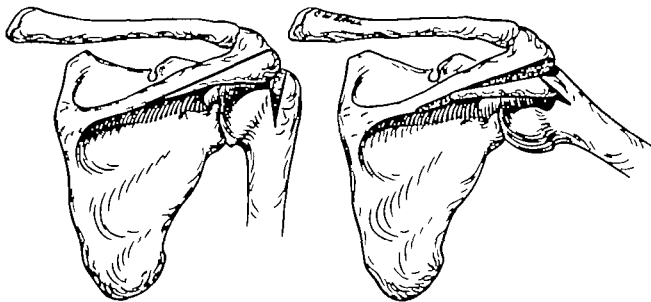


FIG 347 Extra articular arthrodesis (Putti)

icle are then forced downward, so that they engage beneath the bone flap elevated from the humerus when the humerus is abducted (Fig 346)

EXTRA ARTICULAR ARTHRODESIS OF PUTTI

The patient is placed on the unaffected side and the arm is draped independently permitting free manipulation of the extremity. A long skin incision is made along the entire length of the spine of the scapula and continued downward on the lateral aspect of the arm. By sharp subperiosteal dissection all soft tissues are stripped from the spine for its entire length and from the acromion process. Next the spine is detached from its base by a thin osteotome beginning at its vertebral end and proceeding toward the acromion. The acromion is divided longitudinally into medial and lateral segments; the medial segment remains attached to the scapula, while the lateral is continuous with the spine of the scapula. The upper end of the humerus is exposed by splitting the deltoid fibers from above downward.

Some workers sacrifice the axillary nerve and the posterior humeral circumflex artery in this step. However this can be avoided by staying in the safety zone below the acromion. Severance of the nerve following arthrodesis of the glenohumeral joint is of no great significance because the shoulder girdle will now be motivated by the trape-

zius and the serratus anterior muscles (the deltoid no longer playing a role in movements of the arm). Nevertheless it is poor surgical technic to divide a healthy nerve unless it is absolutely necessary. A bone flap is now elevated outward from the lateral surface of the humerus and the bone transplant comprising the acromion and the spine is wedged beneath it so that the acromion fits tightly under the osseous flap. With the arm abducted the transplant is anchored by a stout chromic suture to the raw surface of the medial portion of the acromion (Fig 347).

Intra articular Arthrodesis (Steindler) Most so-called intra-articular procedures are really combined intra-articular and extra articular operations. Steindler's technic however is truly an intra articular arthrodesis. The superior aspect of the glenohumeral joint is exposed through a semicircular incision around and beneath the acromion. The medial flap is reflected from the superior surface of the acromion for an inch. Next the central origin of the deltoid muscle and its acromial attachment is identified and the acromion is divided from $\frac{1}{2}$ to $\frac{3}{4}$ inches from its lateral margin with a thin osteotome. The detached acromion with the deltoid is reflexed downward thus exposing the subacromial bursa and the musculotendinous cuff. A long

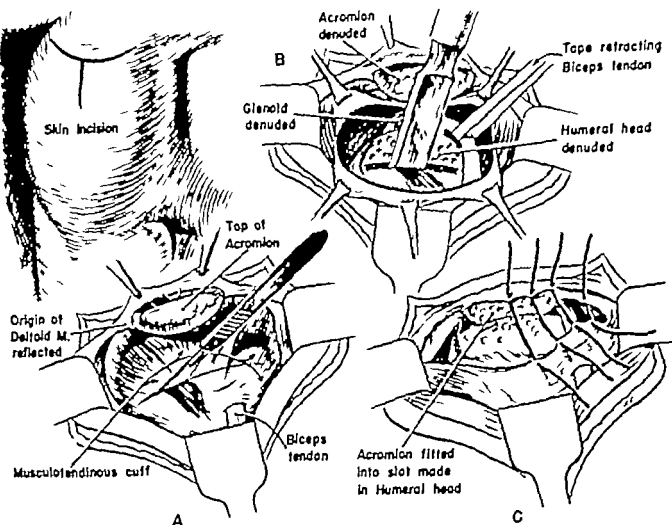


FIG 348 Combined intra articular and extra articular arthrodesis (Gill)

tudinal incision in the capsule provides access to the inside of the joint which is further visualized by traction on the arm. If more exposure is desired a transverse cut may be added to the longitudinal capsular incision at the glenoid margin.

Both the glenoid and the head of the humerus are denuded of all cartilage down to cancellous bone. Two drill holes are made in the base of the acromion and two in the head of the humerus. Stout chromic catgut sutures are passed through the drill holes but not tied. The capsule is closed then the sutures are tied over the capsule. After discarding the detached portion of acromion process the deltoid is reattached posteriorly to the spine of the scapula to prevent backward displacement of the head of the humerus.

Combined Intra articular and Extra articular Arthrodesis (Gill) Gill's opera-

tion is relatively simple. It was designed especially for nontuberculous affections of the shoulder joint. The author has used it in tuberculosis of the shoulder without unfavorable sequelae. Theoretically surgical trauma inflicted by performing an intra articular arthrodesis on a tuberculous joint favors the development of undesirable and serious complications. In practice however this is not true. Therefore Gill's method has been employed in many cases of tuberculous arthritis of the shoulder without fear of incurring distressing complications which, as yet the author has never encountered.

A U incision is made about 2 centimeters below and around the margin of the acromion. Beginning at the center of the incision, a vertical limb is extended from 2 to 2½ inches downward on the lateral aspect of the shoulder. The deltoid is detached from the acromion process by sharp sub-

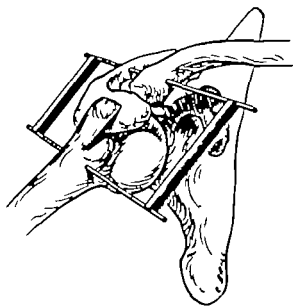


FIG. 349 Method of obtaining compression following arthrodesis of the scapulohumeral joint (author's method)

periosteal dissection and reflected downward exposing the upper end of the humerus the subacromial bursa and the musculotendinous cuff. Also by sharp dissection with a sharp thin osteotome the superior and the inferior surfaces of the acromion are denuded next leaving the periosteum attached proximally on the superior surface.

A longitudinal incision in the capsule exposes the articular surfaces of the glenoid cavity and the humeral head. The upper portion of the capsule is detached from the rim of the glenoid cavity and then together with the tendinous portion of the rotator cuff is excised up to an inch of its insertion into the greater and the lesser tuberosities. A sharp curette readily removes the cartilage from the glenoid cavity and a sharp thin osteotome removes it from the humeral head. A thin osseous flap is elevated upward from the anterolateral surface of the humerus. A wedge-shaped piece of bone with its base upward is removed from the remaining portion of the head. The denuded acromion process fits snugly into the humeral defect upon abducting the humerus. The capsule and cuff attached to the head of the humerus are anchored by stout mattress sutures to the intact periosteum and fascia

on the superior surface of the acromion thus ensuring maintenance of the humeral position (Fig. 348).

Arthrodesis by Compression. The principle of compression has been added by the writer to the operation designed by Gill. Its advantages justify its usage. By the simple method about to be described, the head is held in the desired position in relation to the glenoid, the tendency of the head to be displaced posteriorly is overcome and by maintaining constant contact between the raw bone surfaces, early union is assured.

After the arthrodesing operation is completed the skin is closed, and the arm is placed in the optimum position of function. A stab wound is made 1 centimeter medial to the acromioclavicular articulation on the anterior surface of the clavicle. A small hole is drilled through the entire thickness of the clavicle parallel with the acromioclavicular joint. A Steinmann pin $\frac{7}{64}$ inch in diameter is inserted through this drill hole. The pin is passed posteriorly through the spine of the scapula and the skin on the posterior aspect of the shoulder.

A similar pin is inserted through the head of the humerus from before, backward, below the implant of the acromion into the humeral head parallel with the first pin (Fig. 349). Both pins are incorporated in the plaster cast. After the cast has hardened the ends of the pins are freed by cutting away the plaster of Paris between and around the pins forming a rectangular slot in front and behind (Fig. 350 top). Positive pressure is created between the raw bone surfaces of the scapula and the humerus by stout rubber bands passing around the ends of the pins. Constant compression is maintained for 5 weeks then the pins are removed. Roentgenographic studies usually evidence osseous union by this time.

POSITION OF OPTIMUM FUNCTION

As previously noted many failures of shoulder joint arthrodesis can be attributed to the poor functional position of the humerus in relation to the scapula. In chil-

dren, the position giving optimum function is for girls, 45° abduction 15 forward flexion and from 15 to 20 internal rotation (attained by tilting upward the flexed forearm from 25° to 30° above the horizontal plane) for boys 55° abduction and

exercised to maintain this position while the plaster cast is applied. The plaster spica jacket extends from the metacarpophalangeal joints to the trochanters and is molded over the iliac crests so that it sits firmly on the pelvis. It is suspended on the opposite

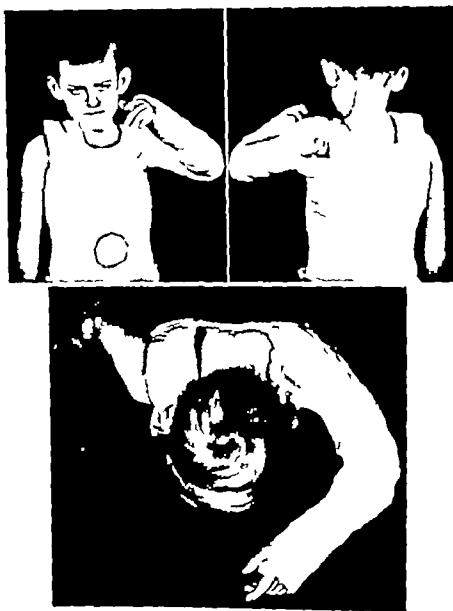


FIG. 350. Position of extremity in plaster after arthrodesis of the scapulohumeral joint. Observe (top right) the slot cut out of the plaster and protruding ends of the pins which are being pulled together by rubber bands. A similar slot is cut out of the front of the cast.

25 forward flexion and the same amount of internal rotation.

After the arthrodesing operation is completed and the humerus is placed in the position of optimum function, care must be

shoulder by a wide plaster strap (Fig. 350).

Generally, without using compression, osseous union is achieved in 8 to 12 weeks in nontuberculous cases. In those employing compression, union occurs in 5 weeks.

However, whether compression is or is not used, immobilization of the shoulder is maintained for a minimum of 12 weeks. In cases not using compression if bony union is not demonstrable by roentgenographic study at the end of this time, another plaster cast is applied for 8 more weeks.

As a rule, in tuberculous shoulders the period of immobilization is longer before firm bony union is attained. Compression in arthrodesis of tuberculous shoulders ensures bony union, decreases the period of immobilization and prevents loss of the desired optimum position of the humerus in relation to the scapula. Rarely is immobilization necessary after 5 months. No abduction splints are used after the plaster is removed.

Removal of the cast is followed by an intensive regimen of active exercises which aim to attain the maximum amount of motion in the extremity by increasing the functional capacity of the muscles of the scapula, the arm and the forearm and to restore normal motion in the elbow and the wrist joints.

MUSCLE TRANSPLANTATIONS

Generally muscle transplants for paralytic disorders of the shoulder joint fail to give satisfactory results. The Research Committee of the American Orthopedic Association studied 62 transplants in 43 patients and observed that the only satisfactory transplantations were in individuals possessing fair power in the deltoid muscles before operation (8 cases). The best results were attained in cases in which a strong posterior deltoid was transplanted anteriorly to the acromion and the outer third of the clavicle (3 cases). The committee questions the value of the Nicola procedure to stabilize paralytic subluxation. A strip of fascia lata was employed in 1 case to tie the scapulae together, in an effort to stabilize the shoulders. No improvement in function was attained by this procedure. It became apparent from the afore mentioned obser-

vations that arthrodesis of the scapulohumeral joint is the operation of choice to achieve optimum function in a paralytic shoulder provided that there is sufficient power in the upper trapezius and the upper two-thirds of the serratus anterior muscles to motivate the scapula.

PARALYSIS OF THE DELTOID MUSCLE

Paralysis of the deltoid most frequently results from acute anterior poliomyelitis. It may be due to such trauma as dislocation of the glenohumeral joint and gunshot wounds in this region. Also it may be caused by obstetric lesions. Careful evaluation of all the muscles of the extremity is essential when contemplating muscle transplantation for a paralyzed deltoid—a procedure justified only when the other muscles particularly the upper trapezius and the pectoralis muscles, have sufficient power to ensure better function.

Muscle transplants are contraindicated in cases with weakened power in the elbow and the hand. Arthrodesis of the scapulohumeral joint in the proper angle of abduction provides more stability and improves function in these cases. Muscle transplants employed to stabilize paralytic dislocations have also proved to be ineffective. The Nicola operation or one of its modifications is the most popular procedure. Here again, arthrodesis of the shoulder joint is the preferred method of stabilization. The most popular muscle transplantations are:

- 1 Extension of the trapezius muscle by virtue of a graft of fascia lata and insertion of the graft into the humerus at the insertion of the deltoid.

- 2 Transference of the biceps and the triceps to the acromion.

- 3 Transference of the posterior portion of the deltoid to the anterior aspect of the acromion and to the outer third of the clavicle, and

- 4 A combination of any of the above methods.

TRANSPLANTATION OF FASCIAL EXTENSION OF THE TRAPEZIUS MUSCLE (MAYER)

This procedure is indicated only when there is good power in the trapezius the pectoralis major and the scapular muscles

particularly the serratus anterior the rhomboid and the levator scapulae. The best functional results are obtained when the deltoid possesses some power.

OPERATIVE TECHNIC

A U skin incision is made around the

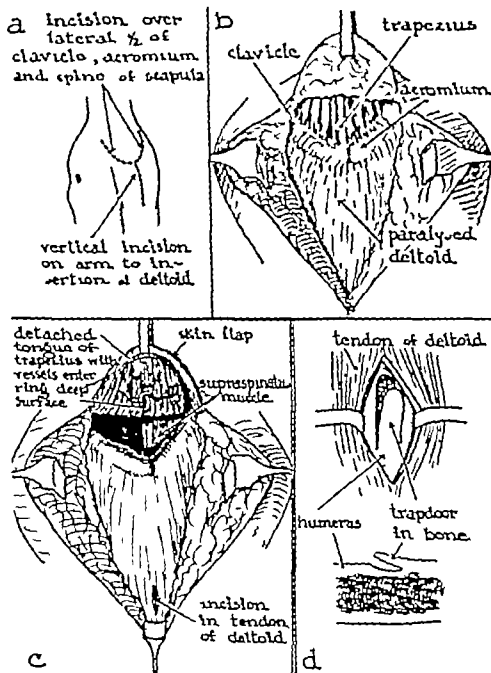


FIG 351A Extension of the insertion of the trapezius muscle to the deltoid tubercle by a graft of fascia lata. (a) Skin incision (b) Skin flaps have been reflected disclosing the underlying trapezius muscle and paralyzed deltoid muscle. (c) Trapezius muscle is detached from the clavicle acromion and spine of the scapula then reflected upward a vertical incision is made in the tendon of the deltoid down to the cortex of the humerus. (d) Trapdoor is made in the lateral aspect of the humerus. (Mayer and Lewis Practice of Surgery vol 3 p 82 Hagerstown Prior)

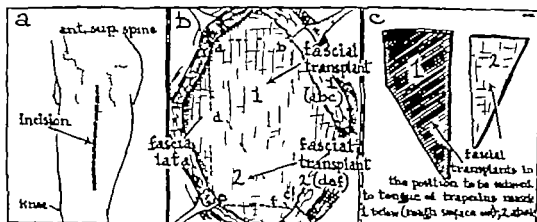


FIG 351B Extension of the insertion of the trapezius muscle to the deltoid tubercle by a graft of fascia lata (*continued*) (a) Longitudinal skin incision on the lateral aspect of the thigh (b and c) A rectangular piece of fascia lata 9 x 4 is removed and divided into a large and small triangular piece corresponding to fascial transplant 1 and 2 (Mayer and Lewis Practice of Surgery Vol 3 p 83, Hagerstown Prior)

shoulder girdle extending along the anterior surface of the clavicle around the acromion and along the spine of the scapula. A second vertical skin incision is made in the lateral surface of the shoulder beginning at the acromion and terminating at the deltoid tubercle of the humerus. The skin on the top of the shoulder is reflected medially exposing the insertion of the trapezius. This is divided and reflected upward from the clavicle the acromion and the spine for from 3 to 4 inches at which point the blood vessels and the nerves penetrate its deep surface.

Anteriorly the plane of dissection lies between the trapezius and the outer border of the sternocleidomastoid muscle posteriorly between the trapezius and the supraspinatus muscle. The anterior and the posterior flaps of the vertical skin incision are dissected away from the top of the deltoid until the entire muscle is exposed. A vertical slit from 1 to 1½ inches down to the bone in the insertion of the deltoid is then made. Through this slit a bone flap is elevated from the lateral aspect of the humerus. A graft of fascia lata from 3½ to 4 inches wide and about 9 inches in length is removed from the lateral aspect of the thigh. The fascial transplantation is divided into a long graft and a short graft as shown in Figure 351. The rough surface of the longer graft

is placed in contact with the deep surface of the trapezius and is anchored by interrupted sutures. The smaller graft covers the outer surface of the muscle and is sutured to both the muscle and the longer graft. The trapezius is now enveloped in two layers of fascia lata.

Next with the arm abducted 135° from the trunk and flexed forward about 20° the edges of the long fascial graft are sutured to the anterior and the posterior borders of the deltoid. Finally the pointed end of the long graft is anchored in the slit beneath the bone flap raised from the humerus.

POSTOPERATIVE MANAGEMENT

A plaster shoulder spica is applied holding the arm in 135° abduction and 20° forward flexion. After 4 weeks graduated active exercises are initiated although the arm is not permitted to drop to the side for 4 more months. An abduction brace may be worn for the remaining period of immobilization after the first 4 weeks.

TRANSPLANTATION OF BICEPS AND TRICEPS MUSCLES (OBER)

The success of this procedure as the transference of the trapezius muscle depends upon good power in the biceps and the triceps muscles and in the pectoralis

major and scapular muscles. The best results are seen in cases with some power in the deltoid.

OPERATIVE TECHNIC

A superior skin incision is made over the acromion process. The anterior limb ex-

tends downward 3 inches over the anterior aspect of the shoulder. The posterior limb is carried downward and slightly inward for 3 inches over the posterior aspect of the shoulder. The interior segment of the incision is deepened through the fibers of the deltoid exposing the coracoid process and

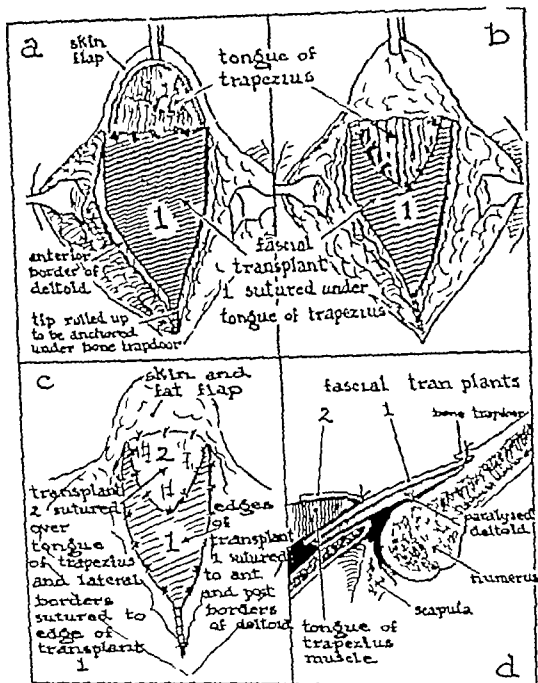


FIG 351C Extension of the insertion of the trapezius muscle to the deltoid tubercle by a graft of fascia lata (*continued*) (a) Fascial transplant 1 is sutured to the undersurface of the trapezius muscle (b) The edges of the tongue of the trapezius are sutured to fascial transplant 1 (c) Fascial transplant 2 is sutured over the tongue of the trapezius and its lateral edges tacked to transplant 1 (d) The top of transplant 2 is rolled up and anchored beneath the trapdoor on the lateral aspect of the humerus, while the arm is held in a position of abduction. (Mayer and Lewis *Practice of Surgery*, Vol 3 p 84 Hagerstown, Prior)

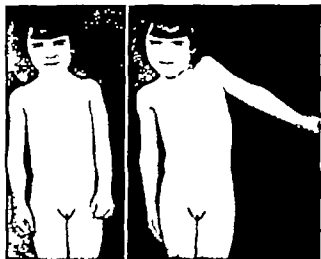


FIG 352 (Above) Patient before transplantation of the biceps and triceps tendons who had limited power in the deltoid muscle. (Right) Patient after operation, showing range of elevation of the arm. (Ober J.A.M.A. 99 2182)

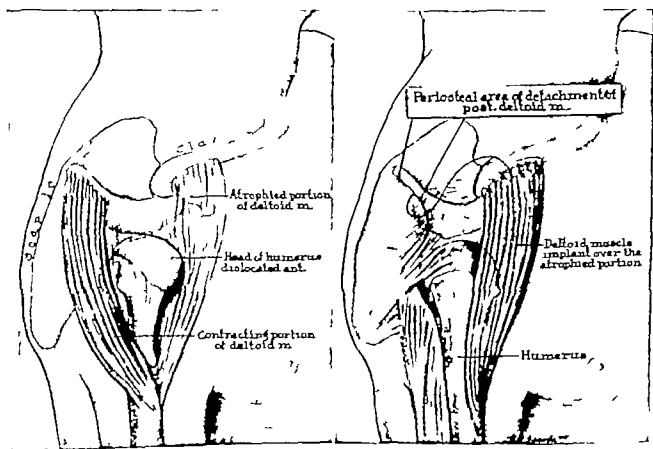


FIG. 353 Transplantation of the origin of the posterior portion of the deltoid muscle to an anterior position. (Harmon P G Surg Gynec. & Obst. 84 117 118)

its muscular attachments. The tendon of the short head of the biceps is identified, dissected free from the coracobrachialis and the pectoralis minor and detached from the coracoid with a small portion of its bony insertion. It is then separated from surrounding structures down to the tendon of the pectoralis major.

If the biceps tendon is too short to reach the top of the acromion, the upper half of the tendon of the pectoralis major may have to be severed to gain length. Next, the posterior arm of the skin incision is developed to expose the long head of the triceps muscle at its origin on the scapula. The tendon with a small portion of its bony attachment is removed from its scapular origin. Together with the attached muscle it is then dissected from the surrounding structures on the upper end of the humerus. With a thin osteotome, two cuts are made in the acromion, one anterior and one posterior for the biceps and the triceps tendons respectively. The cuts are made parallel with the flat surface of the acromion and their edges are spread to receive the tendons. Both tendons are passed through the fibers of the deltoid to emerge near the top of the acromion opposite their respective bone cuts. The tendons are anchored therein with interrupted silk sutures, while the arm is being held in an abducted position. Postoperative management is the same as described for the transplantation of the trapezius muscle.

TRANSPLANTATION OF THE ORIGIN OF THE POSTERIOR PORTION OF THE DELTOID

Occasionally the posterior portion of the deltoid is not implicated while the remaining portions disclose complete paralysis. Ober believes that transferring this segment to an anterior position increases the function of other muscle transplants about the shoulder. However, as previously stated, it appears that the only transplants to function adequately are those performed in indi-

viduals who possess some power in the deltoid. Harmon has employed this procedure to restore power of abduction to the extremity and also to cure recurrent dislocation of the glenohumeral joint associated with paralysis of the anterior and the middle portions of the deltoid.

OPERATIVE TECHNIC (HARMON)

A semicircular incision is made around the shoulder just below the acromion. The incision proceeds along the lower border of the middle third of the clavicle around the acromion, to the middle of the spine of the scapula. The entire line of insertion of the deltoid is exposed by reflecting the skin flaps upward and downward. The scapular origin of the deltoid is freed subperiosteally with a sharp thin osteotome, and the upper half of the muscle is mobilized by separating it from the surrounding structures.

Care must be exercised during this stage of the operation not to traumatize the axillary nerve and the posterior axillary artery as they emerge from the quadrilateral space and continue anteriorly on the deep surface of the muscle. A new site of insertion is now prepared on the anterior surface of the outer third of the clavicle by exposing the bone by sharp subperiosteal dissection. The scapular origin is transferred anteriorly and attached by interrupted sutures to the edge of the periosteum on the superior surface of the clavicle. The extremity is immobilized in a plaster shoulder spica in 105° elevation in the frontal plane. Postoperative management is similar to that described for the method of Mayer and Ober.

FASCIAL TRANSPLANTS IN PARALYTIC DISORDERS ABOUT THE SHOULDER GIRDLE

Muscle imbalance resulting from infantile paralysis in the cervical region and the scapular muscles may be responsible for extensive deformities of the cervicothoracic and the upper thoracic regions of the trunk and the shoulder girdles. Such deformities

are difficult to control and more difficult to correct. Dickson, stimulated by the work of Lowman and of Mayer, designed several fascial transplants which tend to stabilize cervicothoracic curves—also cases with involvement of the scapular muscles.

PARALYSIS OF THE SPINAL AND THE ELEVATOR MUSCLES OF THE SCAPULA

Paralysis or weakness of the spinal and the elevator scapular muscles may lead to severe high cervicothoracic scoliosis and dropping of the shoulder on the affected side. In these cases the goal is to elevate the depressed shoulder and to stabilize the deformity which is progressive in nature by creating a fixator action against the pull of the unaffected muscles on the convexity of the cervical curve. Dickson reports satisfactory results with the following procedure. Moreover the function of the entire upper extremity improves and fatigue and neck pain are lessened.

OPERATIVE TECHNIC (DICKSON)

Two long strips of fascia lata rolled into tubes with the gliding surfaces out are employed in this operation. A skin incision of from 3 to 4 inches is made over the outer extension of the spine of the scapula close to the acromial end; the spine is exposed subperiosteally and a slot is made through it. A second incision is made from the spine to the apex of the cervical curve on the concave side exposing the underlying cervical fascia. The end of one of the fascial tubes is now laced to the cervical muscles at the apex of the curve on its concave side. The dropped scapula is elevated as high as possible, and while this position is being maintained the other end of the fascial tube is passed through the slot in the spine and sutured to itself with interrupted silk sutures.

A third incision is made over the spinous process of the first thoracic vertebra and a hole is drilled through its base. One end

of the second fascial tube is drawn through a slot made in the vertebral end of the spine and anchored to itself. The other end traverses a subcutaneous tunnel, emerging at the spinous process of the first thoracic vertebra; it passes through the hole and is sutured to itself under tension.

POSTOPERATIVE MANAGEMENT

The arm is immobilized in a plaster spica in from 40° to 50° abduction, or the arm is attached to the head of the bed. Active exercises are begun after 3 weeks. All support is discontinued after 5 or 6 weeks.

PARALYSIS OF THE SERRATUS ANTERIOR MUSCLE

Weakness or paralysis of the upper spinal and scapular muscles is responsible for pronounced hypermobility of the scapula and promotes high thoracic and cervicothoracic curves. The serratus anterior functions primarily as a stabilizer of the scapula. In pushing movements it draws the scapula toward the thoracic cage; in elevating it rotates the inferior angle upward and forward. If the serratus anterior is implicated the scapula shifts toward the vertebral column and rotates downward and inward. Marked winging of the scapula occurs during arm elevation. The deformity is particularly striking in the movements of pushing. In the operation about to be described an attempt is made to stabilize the scapula, which in turn improves the shoulder function and also acts as a check to further progression of the spinal curvatures.

OPERATIVE TECHNIC (DICKSON)

First the scapula is displaced forward to the position desired in relation to the thoracic wall. While it is held in this position an incision is made above the lower axillary border of the scapula, and a drill hole is passed through the inferior angle. Through this hole the end of a tube made of fascia lata is passed and sutured to itself.

A second incision is made on the lateral

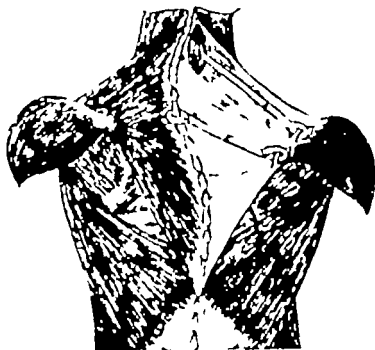
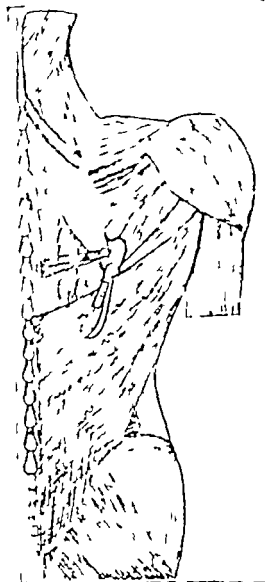


FIG. 354 Fascial transplants designed by Dickson for paralytic disorders about the shoulder joint. (Dickson J Bone & Joint Surg. 19 408)



aspect of the thorax over the origin of the pectoralis major muscle the two skin incisions are connected by a subcutaneous tunnel through which the distal end of the fascial tube is passed. The end of the tube is split into two halves one half is laced into the lowermost fibers of the pectoralis major the other into the anterior border of the latissimus dorsi. Then the ends of the fascial tube are anchored under considerable tension while the scapula is held in the desired position. Added stability of the scapula may be attained by running a second fascial tube from the cervical muscles to the spine of the scapula.

border is pulled downward and inward by the unapposed serratus anterior, in addition to a lateral shift of the scapula. High thoracic scoliosis may be a concomitant finding the deformity tends to be progressive.

OPERATIVE TECHNIC (DICKSON)

Two fascial transplants in the form of tubes are used in this procedure. One extends from the lower vertebral border of the scapula to the spinal muscles the other from the inferior angle of the scapula to the latissimus dorsi muscle. A small incision is made over the lower vertebral border of the scapula, and a hole is drilled through the bone another small incision is made over the spinal muscles opposite and slightly inferior to the first incision. The two incisions

PARALYSIS OF THE RHOMBOIDEI AND THE LEVATOR SCAPULAE MUSCLES WITH GOOD POWER IN THE SERRATUS ANTERIOR MUSCLE

With paralysis or weakness of the levator scapulae and the rhomboides muscles the scapula shifts laterally toward the axilla. During arm elevation its upper vertebral

are connected by a subcutaneous tunnel. One end of a fascial tube is laced into the spinal muscles; the other end traverses the subcutaneous channel, proceeds through the hole in the vertebral border of the scapula and is sutured on itself under tension.

A third incision is made over the inferior angle of the scapula. The bone is exposed subsequently and a hole is drilled through its substance. Another small vertical incision is made 3 inches medial and inferior to the inferior angle of the scapula, exposing the fibers of the latissimus dorsi. Both incisions are also connected by a subcutaneous tunnel. One end of the second fascial tube is laced into the fibers of the latissimus dorsi. The other passes through a tunnel hole in the inferior angle of the scapula and is sutured to itself under tension.

As pointed out by Dickson, the aforementioned procedures are useful in improving the overall function of the extremity. They stabilize the scapula and may arrest partially the development of spinal deformities. More study of the usefulness and the indications of fascial transplants is necessary before definite conclusions can be drawn as to the merit of the procedures.

AMPUTATIONS

GENERAL CONSIDERATIONS

Every effort should be made to save as much of the upper arm as possible when amputation through this region is contemplated. A stump of less than 4 inches is of no functional value because it is impossible to fit a workable prosthesis on such a short stump. On the other hand, the upper end of the humerus should be preserved if possible even if the desired stump length is not attainable since the normal contour of the shoulder girdle is retained. Removal of the head of the humerus produces a narrow flat shoulder.

The most desirable amputation incision through the upper end of the humerus is the racquet incision which creates a long lateral and a short medial flap. In this method the lateral flap consisting of the deltoid

muscle is utilized to cover the end of the humeral stump.

Disarticulation of the shoulder is usually performed for severe traumatic injuries or malignant tumors implicating the upper arm. When feasible, amputation through the upper arm is preferable to disarticulation. As in amputation through the surgical neck of the humerus, the racquet incision provides both adequate exposure for rapid disarticulation and good flaps for easy closure.

Interscapulothoracic amputation is indicated for malignant lesions of the shoulder girdle, particularly when the component elements of the glenohumeral joint are implicated. If the tumor is limited to the clavicle or the scapula, resection of these bones should be performed. Loss of great amounts of blood may be minimized by first isolating and ligating the subclavian artery and vein.

AMPUTATION AT THE LEVEL OF THE SURGICAL NECK

The patient is placed in a sitting position with the shoulder over the side of the table, a sandbag under the shoulder and the arm draped separately. The table is tilted about 30° away from the operator. The handle of the racquet incision begins on the anterior surface of the arm at the lower border of the clavicle in the interval between the deltoid and the pectoralis major muscles. It continues downward along the anterior border of the deltoid to the level of the deltoid tubercle; thence it curves outward to and continues along the posterior border of the deltoid to the posterior fold of the axilla.

Beginning at the lower end of the handle of the racquet incision, the medial incision is carried around the inner aspect of the arm an inch below the axilla and meets the lateral incision. The insertion of the pectoralis major muscle is divided and allowed to retract medially. Next the axillary vessels and nerve trunks are exposed as they pass from beneath the pectoralis minor muscle medial to the coracobrachialis muscle. The artery and the vein are isolated doubly ligated and divided. The nerve

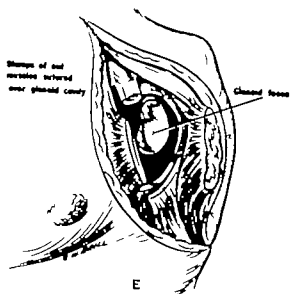
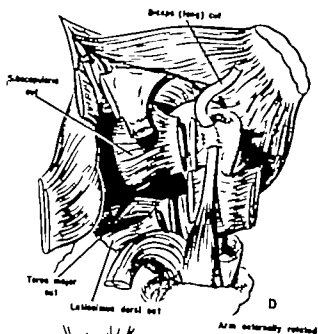
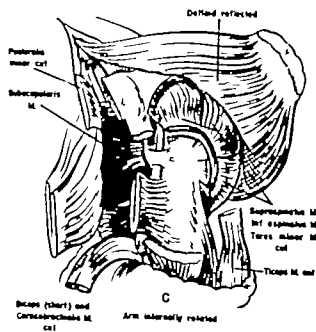
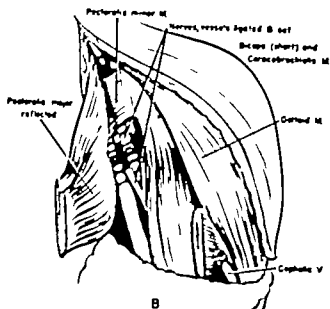
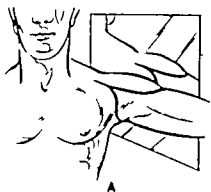


FIG 355 Technic of disarticulation of the shoulder

trunks (radial, median and ulnar nerves) are isolated, ligated and severed after being injected first with procaine, then with 75 per cent alcohol

No traction is made on the trunks before cutting them. The tendinous insertion of the deltoid is then divided, and the muscle, together with the skin flap, is reflected upward. Care is taken not to injure the axil-

lary nerve. The tendinous insertions of the latissimus dorsi and the teres major muscles are cut on the inner aspect of the humerus. The long muscles running parallel with the shaft of the humerus (biceps triceps and coracobrachialis) are severed approximately one inch below the contemplated level of the stump of the humerus. Next, the humerus is sectioned through the surgical neck. The deltoid muscle is approximated and sutured to the muscles on the inner aspect of the stump of the humerus, and the skin edges are brought together with interrupted sutures.

DISARTICULATION OF THE SHOULDER

The patient is placed in the sitting position with the shoulder over the side of the table, a sandbag under the shoulder and the arm draped separately to allow easy maneuvering. The table is tilted 30° away from the surgeon.

The vertical arm of the racquet incision begins on the anterior aspect of the arm

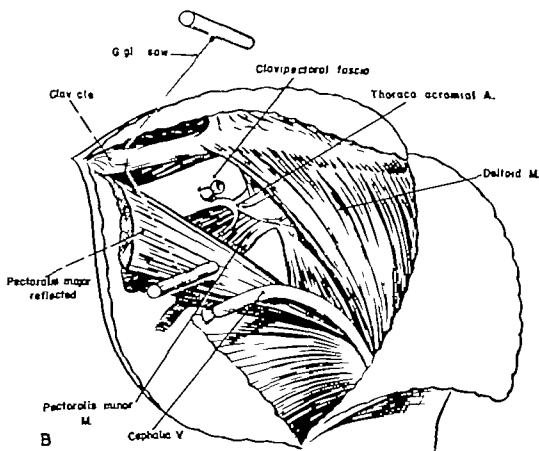
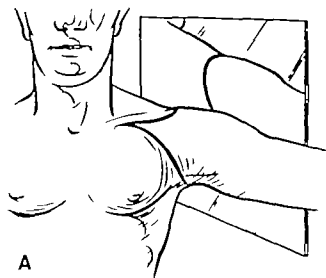


FIG. 356 Technic of interscapulothoracic amputation.

just below the lower border of the clavicle in the sulcus between the pectoralis major and the deltoid muscles. It continues downward along the anterior border of the deltoid

to the level of the deltoid tubercle then sweeps outward and continues along the posterior border of the deltoid to the posterior axillary fold. A second transverse in-

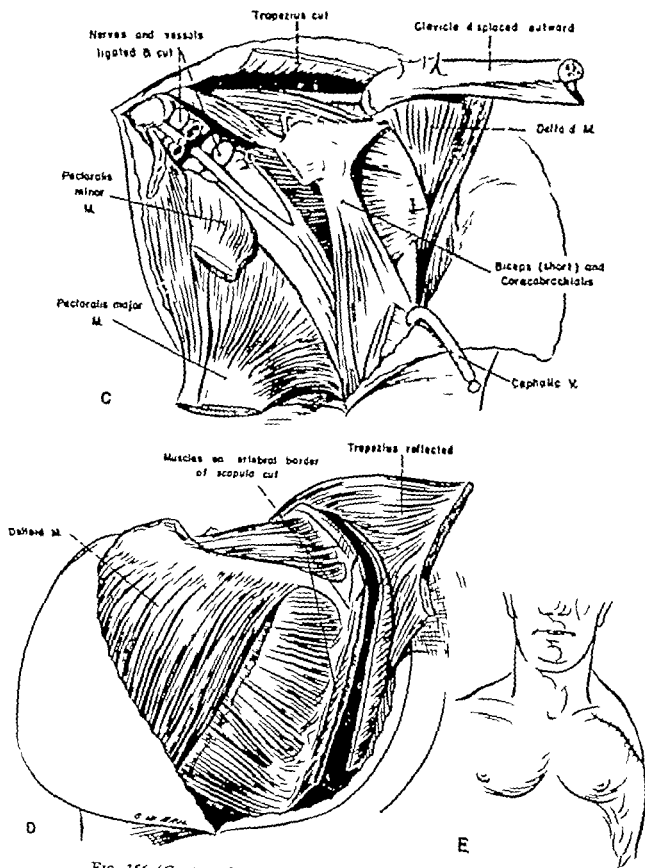


FIG 356 (Continued) Technic of interscapulothoracic amputation

cision is made, passing through the axilla connecting the two ends of the first incision. The anterior arm of the incision is deepened, exposing the pectoralis major muscle, which is divided close to its insertion into the humerus. Next, the sulcus between the coracobrachialis and the pectoralis minor muscles is developed exposing the axillary vessels and the nerve trunks. The vessels are separated doubly ligated and divided between ligatures. The nerve trunks are ligated, injected with procaine then with 75 per cent alcohol and severed.

The conjoined tendon of the coracobrachialis and the short head of the biceps muscle are cut at their insertion into the coracoid process. The deltoid is divided at its point of insertion into the humerus then it is reflected upward together with the skin flap off the anterior surface of the humerus bringing into view the upper end of the humerus and of the musculotendinous cuff. External rotation of the arm puts the subscapularis tendon on a stretch and together with the capsule it is cut close to its insertion into the lesser tuberosity. Then the arm is rotated internally. The tendon of the long head of the biceps muscle the remainder of the musculotendinous cuff and the posterior portion of the capsule are divided close to its insertion into the greater tuberosity. By pulling downward on the arm the inferior capsule is visualized and cut.

Disarticulation is completed by dividing the remaining muscles on the anterior (teres major and latissimus dorsi muscle) and posterior (triceps muscle) aspects of the humerus. All stumps of the severed muscles are sutured across the glenoid cavity. The lateral flap is approximated to the medial flap and the skin edges are brought together by interrupted sutures. Drainage of the wound for the first 24 hours is advisable because of the amount of secretion that follows this procedure. Dry dressings are applied to obliterate all dead spaces. After 24 hours the drain is removed and a compression dressing is applied (Fig. 355).

INTERSCAPULOTHORACIC AMPUTATION

The patient is placed in a sitting position with the shoulder well over the side of the table, a sandbag under the shoulder, and the arm draped separately to permit easy maneuvering of the limb. The head is drawn in the opposite direction. The table is tilted 30° away from the operator thereby making both the front and the back regions of the shoulder accessible. A linear skin incision is made on the anterior surface of the clavicle beginning at the outer border of the sternocleidomastoid muscle and carried outward crossing the acromioclavicular joint.

A second incision starts at the lower border of the center of the clavicle and continues downward in the interval between the deltoid and the pectoralis major to the lower border of the anterior axillary fold. Before making the axillary and the posterior portions of the skin incision, the clavicular incision is carried down to the bone and the clavicular portion of the pectoralis major muscle is stripped subperiosteally from its bony attachment. The clavicle is then divided with a Gigli saw at the junction of its middle and inner thirds. The outer segment of the clavicle is freed from all soft tissue attachments and is removed by cutting it free at the acromioclavicular joint.

The interval between the pectoralis major and the deltoid muscles is developed exposing the coracoid process. The pectoralis major is divided close to its insertion into the humerus and is allowed to retract medially. The pectoralis minor is severed at the tip of the coracoid process. By developing the interval between the pectoralis minor and the coracobrachialis muscles the large vessels and the nerve trunks are brought into view. The subclavian artery and vein are separated doubly ligated and cut between ligatures. The nerve trunks are freed injected with procaine then with 75 per cent alcohol and divided. Next the latissimus dorsi is divided and the limb falls outward from the trunk.

The clavicular incision is now extended backward over the shoulder along the vertebral border of the scapula to the inferior angle then sweeps outward to the posterior axillary fold. The arm is raised, and the anterior incision is continued across the axilla to meet the posterior incision. With the arm carried across the chest the posterior flap is reflected to expose the trapezius muscle which is detached from the scapula. All muscles running from the thorax to the vertebral border of the scapula are severed from above downward, and the limb is removed with complete severance of all muscular attachments to the scapula.

All muscle stumps particularly those of the pectoralis major and the trapezius are sutured to the chest wall. Bleeding vessels are ligated the flaps are tailored and the wound is closed with interrupted sutures. In most cases a drain is advisable in the dependent portion of the wound for 1 or 2 days. Dry dressings are firmly applied to eliminate dead tissue spaces (Fig 356).

RESECTIONS

EXCISION OF THE ACROMION PROCESS

Hitherto the most common cause for shoulder pains associated with arm movements particularly abduction was believed to be impingement of the supraspinatus tendon against the acromion process. Although the above mechanism is responsible in some cases the study described in the chapter on bicipital syndromes leads one to conclude that the most common etiologic factor for painful shoulders is bicipital tenosynovitis. Nevertheless it must be admitted that in a small group of patients friction and impingement of the supraspinatus tendon against the acromion process will cause pain on abduction of the arm. The symptom complex is referred to as the supraspinatus syndrome.

Normally when the arm is abducted the greater tuberosity readily glides under the acromion or the coraco-acromial ligament with minimum friction and no impingement.

However in the presence of certain alterations in the musculotendinous cuff the subacromial bursa and greater tuberosity these structures actually impinge against the acromion or the coraco-acromial ligament, causing pain on abduction of the arm. Pain is felt as these structures pass under the coraco-acromial arch once they have passed under the arch abduction is completed without pain. The painful arc of abduction is usually between 80° and 120° .

Following acute incomplete or complete tears of the musculotendinous cuff the disrupted tendinous tissue is swollen edematous, inflamed and thickened. Therefore it is apparent that such tissue will be compressed readily against the acromion during abduction of the arm and produce a painful arc of movement. When the acute inflammatory process has subsided and the scar tissue contracts the torn portions of the cuff shrink in thickness. It will now pass under the acromion with minimum friction and without impingement or pain on abduction of the extremity.

Old incomplete tears of the cuff in themselves do not give rise to painful motion. It has been demonstrated in the chapter on degenerative lesions of the shoulder joint that in many cases exhibiting such lesions the cuff still attached to the head of the humerus may be thinned, frayed, shredded or absent, while the torn fibers on the proximal side of the tear may be thickened and hypertrophied. Nevertheless these alterations may allow free painless movements at the shoulder joint.

In such instances, there are often secondary hyperplastic inflammatory changes in the walls of the subacromial bursa which may become so thickened that the bursal sac impinges against the acromion when the arm is abducted thereby causing pain. Increased friction and impingement against the acromion favor more pronounced inflammatory changes in the bursal walls establishing a vicious cycle.

Also, complete and incomplete tears are often associated with the formation of bony

excrecences over the greater tuberosity which impinge against the acromion on arm abduction. Bony excrecences and a hypertrophied subacromial bursa are often concomitant lesions in the same shoulder. Occasionally, calcareous masses within the substance of the cuff may impinge against the acromion. Extensive ossification may occur in the musculotendinous cuff. Impingement of the ossified tissue against the acromion may result in pain and restricted motion.

Most individuals with acute injuries to the musculotendinous cuff recover either spontaneously or after a period of rest and physical therapy. When the causative factors of pain are impingement against the acromion of a thickened bursal sac or bony excrecences, pain is alleviated by excising the bursa or the bony excrecences. In addition, all or part of the acromion should be resected to prevent their reformation. Likewise, in cases with ossification within the cuff, in which excision of the ossified mass is not practical, resection of the acromion is the procedure of choice. In all instances of repaired rupture of the musculotendinous cuff, the acromion should be resected. This added procedure not only facilitates the operation but removes any source of friction and impingement on the reattached cuff.

Smith Petersen points out that acromioplasty relieves pain and muscle spasm and increases shoulder girdle motion in patients afflicted with atrophic arthritis. Although the operation does not increase motion materially in the glenohumeral joint, it does alleviate pain and muscle spasm, thereby increasing motion in the thoracoscapular joint—hence improving the overall usefulness of the extremity. He advocates performing the operation for subacromial bursitis, a concomitant lesion of atrophic arthritis of the shoulder, before osseous changes are roentgenographically demonstrable.

Operative Technic. The patient is placed in a sitting position, with the shoulder well

over the side of the table and a sandbag under the shoulder. The table is tilted 30° from the operator, and the arm is draped separately.

A short saber incision is made just to the outer side of the acromioclavicular joint, beginning at the anterior border of the acromion and extending backward across the acromion to its posterior border. The incision is deepened to the bone, and the central deltoid muscle fibers are detached subperiosteally from the acromion with a sharp osteotome and reflected laterally. The acromion is divided approximately 1 centimeter to the outer side of the acromioclavicular joint in an anteroposterior direction with an osteotome.

Excision of the acromion exposes the roof of the subacromial bursa. As a rule, the bursal walls are thickened and disclose evidence of a chronic inflammatory process. As complete a synovectomy as possible is performed. By retracting the edges of the wound and rotating the arm internally and externally, the entire musculotendinous cuff can be inspected thoroughly. Closure is achieved by suturing the detached deltoid muscle to the periosteum on the top of the acromion.

The entire acromion may be removed without impairing the stability of the acromioclavicular joint, so long as the coracoclavicular ligaments remain intact. The plane of division of the acromion traverses the acromioclavicular joint, thereby severing the acromioclavicular ligaments. If the entire acromion has been resected, the deltoid muscle is sutured to the acromioclavicular ligaments and to the reflected periosteum. The skin edges are approximated by interrupted sutures and a dry dressing is applied, making firm pressure over the wound.

Postoperative Management. A sling provides sufficient immobilization of the extremity. Active motion at the elbow, wrist, and the hand is encouraged immediately. However, active shoulder motion is not permitted until the postoperative reac-

tion about the shoulder recedes. Pendulum exercises are begun after the fourth or fifth day and active abduction of the extremity against gravity is not begun until after the tenth or twelfth day. By this time, active contraction of the deltoid will not elicit pain. Optimum restoration of function should be attained in from 6 to 8 weeks.

RESECTION OF THE SCAPULA

Coley points out that capulectomy is justifiable as an alternative to interscapulothoracic amputation in malignant lesions of the scapula where the entire tumor can be removed. Also such benign lesions as chondromas, which cannot be successfully removed by partial excision may be removed by resection of the scapula. Localized destructive lesions such as chronic osteomyelitis and tuberculosis and benign tumors such as osteochondromas may be dealt with by partial resection of the scapula.

Operative Technic of Total Resection. The patient is placed on the unaffected side close to the edge of the table. The arm is draped separately. Two skin incisions are made. The first begins just lateral to the tip of the acromion process and extends along the spine to the vertebral border of the scapula. The second incision is a continuation of the first downward along the vertebral border to the inferior angle of the scapula. The resulting flaps are reflected exposing the dorsal surface of the scapula. The horizontal limb of the incision is deepened to the bone thereby severing the *tendinous insertions* of the trapezius and the deltoid muscles which are next detached from the acromion process and the spine of the scapula and retracted. The deltoid is also detached from the outer third of the clavicle to bring into view the coracoid process with its muscular attachments, the coracoclavicular ligaments and the insertion of the rotator muscles.

The acromioclavicular ligaments are severed and the scapula is mobilized further by dividing the muscular attachments to its

vertebral border: the levator scapulae, the rhomboids, the serratus magnus and the teres major muscle. Then the muscular attachments to the coracoid process, the coracoclavicular ligament and the coracoclavicular ligaments are divided.

Finally by a curved incision around the upper end of the humerus the four short rotator tendons are divided from before backward. This sweeping incision will also sever the coracohumeral ligaments and the tendon of the long head of the biceps brachii muscle. Re-union of the bone is completed by cutting the remaining portion of the fibrous joint capsule and the muscular attachments. After all bleeding has been controlled the trapezius and the deltoid muscles are approximated and the skin incisions are closed with interrupted sutures. A drain is placed in the dependent part of the wound, it is removed after 24 or 48 hours. Dry dressings are applied, making firm pressure on the wound. The arm is immobilized by a Velpau bandage.

POSTOPERATIVE MANAGEMENT. Early active motion is encouraged in the joints distal to the shoulder. Although shoulder function is greatly impaired by this procedure some useful function is retained in the upper extremity and the operation is by far more desirable than interscapulothoracic amputation when feasible.

Operative Technic of Subperiosteal (Partial or Total) Resection of Scapula (Ollier). Varying amounts of the body of the scapula or the entire bone may be excised by this method. Two skin incisions are made. One begins at the tip of the acromion and extends along the spine of the scapula to its posterior border. The other starts at the posterosuperior angle of the scapula, continues along the vertebral border and ends at the inferior angle. By sharp subperiosteal dissection the muscular attachments (trapezius and deltoid muscles) are reflected from the spine and the acromion process. The periosteum along the posterior border in the interval between the infraspinatus muscle and the insertion of the

rhomboid major is divided and reflected from the infraspinous fossa.

The superior border of the scapula is exposed in a similar manner, and the periosteum is elevated from the supraspinous fossa. Care must be exercised at this point not to traumatize or cut the suprascapular vessels and nerve as they enter the suprascapular notch. Next the inferior angle is raised and the subscapularis muscle is stripped subperiosteally from the subscapular fossa. The posterior border is detached completely from any muscular attachments.

Subperiosteal stripping on the ventral surface of the scapula can now proceed as far as the anterior surface of the neck of the scapula. Any portion of the scapula can be removed with this exposure. The bone may be divided below the base of the spine, or through the neck of the scapula, or the entire scapula can be removed by subperiosteal stripping of all the ligaments attached to the neck.

RESECTION OF THE OUTER END OF THE CLAVICLE

This procedure may be performed for chronic subluxation or hypertrophic arthritis of the acromioclavicular joint. It is especially indicated in older individuals and in those who do not perform strenuous work. The operation gives a very good functional result, alleviates pain, reduces the period of disability and improves the cosmetic effect of the shoulder. However, most individuals who undergo the operation believe that the shoulder is not so strong as the unaffected one. The resection is not performed for complete dislocations of the joint in which the coraco-acromial ligaments are disrupted. If a resection of the outer end of the clavicle is done in these cases, reconstruction of the coraco-acromial ligaments should supplement the procedure.

Operative Technic (Gurd and Mumford). A small curved incision is made over the acromioclavicular joint. The distal end of the clavicle is exposed subperiosteally and raised with bone forceps and brought

forward, while a Gigli saw is passed beneath it. The distal 1 or 1½ inches of the clavicle is sawed off and removed. The meniscus, if present, is excised.

Finally, the periosteal tube is reefed and closed, covering the cut surface of the clavicle. The skin edges are approximated with interrupted sutures and the arm is immobilized in a Velpeau bandage. Active use of the arm is encouraged after a week. Full restoration of shoulder motion should be complete in from 4 to 6 weeks.

RESECTION OF THE INNER END OF THE CLAVICLE

This procedure may be employed in chronic dislocations of the sternoclavicular joint which are causing shoulder dysfunction but in which reconstructive measures are contraindicated or in cases in which reconstructive procedures have failed. Occasionally pronounced degenerative lesions of the sternoclavicular joints are encountered, producing pain and disability. Here resection is a justifiable procedure.

Operative Technic. The sternoclavicular joint is exposed through a 2 to 3 inch incision. The sternal end of the clavicle is exposed subperiosteally, cut with a Gigli saw and removed. The periosteal tube is plicated and closed over the raw end of the clavicle. The skin wound is closed with interrupted suture and the arm is immobilized in a Velpeau dressing. Active use of the shoulder is encouraged after a week. Full function should be achieved in from 4 to 6 weeks.

TOTAL EXCISION OF CLAVICLE

Fortunately the indications for total excision of the clavicle are few, since the close proximity of many vital structures make this a formidable procedure. Tumors of low grade malignancy, giant-cell tumors, benign tumors (which cannot be dealt with by subtotal excision) and necrosis of the clavicle, as seen in chronic osteomyelitis and tuberculosis, are the chief indications for total excision. Tumors may distort the normal

anatomic arm relationship and great difficulty may be encountered in excising the bone without injury to surrounding structures. Then, too, greater danger exists in excising the middle and inner third of the clavicle than the outer third. Whenever possible the plane of dissection should hug the bone closely. The structures more vulnerable to injury are the subclavian vessels, the cephalic vein, the innominate vein on the right, the external jugular vein, the thoracic vein on the left, the pleura and the brachial plexus.

Operative Technique. A horizontal skin incision is made in the long axis of the bone from the sternal to the acromial end. If the operation is for the removal of a tumor the skin flaps are dissected from the tumor and retracted. The attachments of the deltoid and the trapezius muscles are divided. Excision is facilitated by dividing the clavicle and removing the two segments separately or severing the acromioclavicular and the coracoclavicular ligaments thereby mobilizing the acromial end of the clavicle. The outer end is then raised anteriorly. Together with the tumor mass it is dissected carefully from the surrounding structures. Lastly the sternoclavicular ligaments are severed and the clavicle is removed.

Loss of part of or all the clavicle does not seriously impair shoulder function nor does it cause a forward droop of the shoulder.

RESECTION OF THE UPPER END OF THE HUMERUS

It may be necessary to resect the upper end of the humerus for gunshot injuries, giant-cell tumors which exhibit malignant transformation or low grade malignant neoplasms. Occasionally following fracture dislocation of the humerus the head fragment must be removed, either because of severe comminution or complete disruption of its blood supply.

Resection for Neoplasms and Fibular Transplantation. The upper end of the humerus is best exposed by the Cubbins or Henry's incision, the latter being preferred

by the author. The anterior limb of the incision extends along the anterior border of the deltoid muscle, beginning at the lower border of the clavicle over the deltopectoral sulcus and ending at the level of the deltoid tubercle. The horizontal limb is a continuation of the upper end of the anterior incision along the outer third of the clavicle extending backward over the tip of the acromion to the spine of the scapula. After the plane between the deltoid and the pectoralis major muscle is developed the origin of the deltoid is detached from the clavicle and the acromion process with a sharp osteotome and is reflected downward and outward exposing the upper end of the humerus.

A vertical incision is then made in the musculotendinous cuff preferably in the interval between the subscapularis and supraspinatus muscles. The rotator muscles are severed close to their insertion into the greater and the lesser tuberosities, the biceps tendon is left intact and displaced inward and the inner aspect of the upper end of the humerus is freed of all muscular attachments.

Finally, the inferior portion of the capsule is cut from the neck of the humerus and the lateral head of the triceps muscle is detached from the humerus by sharp dissection care being exercised to avoid injury to the musculospiral nerve as it swings around the upper end of the bone. The upper end of the humerus then is projected through the wound and severed several inches distal to the neoplasm through good bone.

The upper end of the fibula may be utilized to restore the length of the humerus and to provide some stability to the shoulder joint. The articular surface of the fibular head is placed in opposition to the glenoid cavity and its distal end is cut to fit into a notch made in the lateral aspect of the shaft of the humerus. The ends of the bones are fixed by two screws, or else the distal end of the fibular graft is inserted into the medullary cavity of the humerus. No

attempt is made to restore the function of the short rotator muscles. The deltoid is reattached to the clavicle and the acromion process, and the skin is closed with interrupted sutures.

The extremity is immobilized for 12

of the humerus is extracted from its dislocated site from above.

An anteroposterior incision is made over the top of the shoulder, just lateral to the acromioclavicular joint, extending from the posterior border of the acromion to a point



FIG 357 (Left) Giant-cell tumor of the left humerus: the tumor and the upper end of the humerus were resected and replaced by a fibular transplant as shown in the center frame. (Center) Fibular transplant of case shown at left, 29 months after operation. (Right) Fibular transplant done for a chondrosarcoma of the upper end of the humerus. A Kirschner wire has been passed through the medullary canal of the fibula as a prophylactic measure against possible fracture of the graft. (J. B. Moore)

weeks in a plaster-of-Paris shoulder spica, with the arm held in abduction of from 45° to 60°. After this interval the arm portion of the spica is bivalved to permit physical therapy and active motion of the elbow and the wrist. The cast is removed after 4 more weeks, but the arm is protected with an abduction brace for several months until bone healing is roentgenographically demonstrable.

RESECTION OF THE HUMERAL HEAD

McLaughlin's transacromial incision provides adequate exposure of the upper end of the humerus, allowing this operation to be performed without danger of injury to the brachial vessels and nerves when the head

2 inches below the anterior border of the acromion process. The incision is carried to the bone and the acromion is divided in a line midway between the acromioclavicular joint and the top of the acromion. By retracting the outer fragment of the acromion and the attached deltoid muscle laterally and downward the subacromial region is visualized. After locating the displaced humeral head one expires it gently from above. The attachments of the musculotendinous cuff and the inferior portion of the capsule are severed from the tuberosities and the neck of the humerus and the upper end of the bone is rounded off with bone-biting forceps. The cuff and the capsule are transplanted at a lower level completely

enveloping the shaft. This step is necessary in order to shorten the rotator muscles thereby compensating for the loss of humeral length. The detached portion of the acromion is discarded and the tendinous insertion of the central deltoid fibers is sutured to a more mesial position on the superior surface of the acromion, thus increasing the efficiency of the muscle as an elevator of the arm. The skin wound is closed with interrupted sutures and the arm

in balanced suspension (Figs. 261 and 262).

Postoperative Management. Active motion is encouraged immediately. Graduated stooping exercises are begun after from 7 to 10 days. Restoration of muscle tone and power is essential in the postoperative regimen. With careful management considerable control of the extremity and a surprisingly good range of painless motion may be obtained. Active motion executed religiously is the key to optimum function.

BIBLIOGRAPHY

- Albee F. H. *Orthopaedic and Reconstructive Surgery*. Philadelphia: Saunders, 1919, pp. 272-6.
- Barr J. S., Freiberg J. A., Colonna P. C., and Pemberton P. A. A survey of end results on stabilization of the paralytic shoulder: report of the research committee of the American Orthopaedic Association. *J. Bone & Joint Surg.* 24: 699, 1942.
- Bosworth D. M. An analysis of twenty-eight consecutive cases of incapacitating shoulder lesions radically explored and repaired. *J. Bone & Joint Surg.* 22: 369-392, 1940.
- Brett, A. L. A new method of arthrodesis of the shoulder joint incorporating the control of the scapula. *J. Bone & Joint Surg.* 15: 969, 1933.
- Cubbins W. R., Callahan J. J. and Scuderi C. S. The reduction of old or irreducible dislocations of the shoulder joint. *Surg. Gynec. & Obst.* 58: 128, 1934.
- Dickson F. D. Fascial transplants in paralytic and other conditions. *J. Bone & Joint Surg.* 19: 405, 1937.
- Dickson, F. D. The treatment of cerebral spastic paralysis with special reference to the Stoffel operation. *J.A.M.A.* 83: 1236, 1924.
- Downman C. E. and Hoke M. The treatment of spastic paralysis. *Arch. Surg.* 9: 135, 1924.
- Fowler E. B. Rupture of the spinati tendons and capsule repaired by a new operation. *Illinois M. J.* 61: 312-334, 1932.
- Gallie, W. E. Tendon fixation in infantile paralysis: a review of one hundred and fifty operations. *Am. J. Orthop. Surg.* 14: 18, 1916.
- Gill, A. B. Surgery of spastic paralysis. *Ann. Surg.* 67: 529, 1918.
- Gill A. B. Stoffel's operation for spastic paralysis. *J. Orthop. Surg.* 3: 52, 1921.
- Gill, A. B. A new operation for arthrodesis of the shoulder. *J. Bone & Joint Surg.* 13: 287, 1931.
- Green W. T. Tendon transplantation of the flexor carpi ulnaris for pronation flexion deformity of the wrist. *Surg. Gynec. & Obst.* 75: 33, 1942.
- Green W. T., and McDermott L. J. Operative treatment of cerebral palsy of spastic type. *J.A.M.A.* 118: 434, 1942.
- Haas, S. L. The treatment of permanent paralysis of the deltoid muscle. *J.A.M.A.* 104: 99, 1935.
- Harmon I. H. Anterior transplantation of the posterior deltoid for shoulder palsy and dislocation in poliomyelitis. *Surg., Gynec. & Obst.* 84: 117, 1947.
- Henry A. K. An operation for slinging a dropped shoulder. *Brit. J. Surg.* 15: 95, 1921, 1928.
- Henry A. K. Exposure of the long bones and other surgical methods. *Bristol Wright*, 1921.
- Hendrick, J. J. Changes in the upper humeral epiphysis following operations for obstetrical paralysis. *J. Bone & Joint Surg.* 19: 473, 1937.
- Kleinberg S. Reattachment of the capsule and external rotators of the shoulder for obstetrical paralysis. *J.A.M.A.* 98: 294, 1932.
- Mayer L. The physiological method of tendon transplantation. *Surg. Gynec. & Obst.* 22: 182, 298, 472, 1916.
- Mayer L. Transplantation of the trapezius for paralysis of the abductors of the arm. *J. Bone & Joint Surg.* 9: 412, 1927.
- Mayer L. Tendon transference of trapezius muscle for paralysis of deltoid muscle. *J. Bone & Joint Surg.* 9: 412, 1927.
- Mayer L. Rupture of the supraspinatus tendon. *J. Bone & Joint Surg.* 19: 640-642, 1937.
- Mayer L. *Tendons, Ganglia, Muscles, Fascia*, in Dean Lewis, *Practice of Surgery*. Hagerstown, Md. Prior, 1942, vol. 3.
- McCarroll, H. R. and Schwartzmann J. P. Spastic paralysis and allied disorders. *J. Bone & Joint Surg.* 25: 745, 1943.
- McLaughlin H. L. Lesions of the musculotendinous cuff of the shoulder: the exposure

- and treatment of tears with retraction, *J Bone & Joint Surg* 26 31 1944
- McLaughlin, H. L. Lesions of the musculo-tendinous cuff of the shoulder differential diagnosis of rupture, *J.A.M.A.* 128 563 1945
- Meyer A. W. Absence of the tendon of the long head of the biceps *J Anat. & Phys* 48 133 1913 1914
- Meyer A. W. Anatomical specimens of unusual clinical interest, *Am. J Orthop Surg* 13 86 1915
- Meyer A. W. Unrecognized occupational destruction of the tendon of the long head of the biceps brachii *Arch. Surg* 2 130 1921
- Meyer A. W. Further observation upon use destruction in joints *J Bone & Joint Surg* 4 491 1922
- Meyer A. W. Evidences of attrition in the human body *Proc. Am. Assn. Anat. Rec.* 27 211 1924
- Meyer A. W. Further evidences of attrition in the human body *Am J Anat.* 34 241 1924
- Meyer A. W. Spontaneous dislocation and destruction of tendon of the long head of biceps brachii, *Arch. Surg* 13 109 1926
- Ober F R. An operation for relief of paralysis of the gluteus maximus muscle *J.A.M.A.* 88 1063 1927
- Ober F R. An operation to relieve paralysis of the deltoid muscle *J.A.M.A.* 99 2182 1932
- Ober F R. Transplantation to improve the function of the shoulder joint and extensor function of the elbow joint, *Am. Acad. Orthop Surgeons Reconstruction surgery of the extremities*, Ann Arbor Edwards 1944
- Ober F R. and Barr J S Brachioradialis muscle transposition for triceps weakness, *Surg Gynec. & Obst.* 67 105 107 1938
- Phelps, W M Treatment of paralytic disorders exclusive of poliomyelitis as Bancroft, F W and Murray C R *Surgical Treatment of the Motor-skeletal System*, Philadelphia Lippincott 1945 vol. 1
- Platt, H. Opening remarks on birth paralysis *J Orthop Surg* 2 272 1920
- Rogers M. H. An operation for the correction of the deformity due to obstetrical paralysis *Boston M. & S J* 174 163 1916
- Royle, N D Treatment of spastic paralysis by sympathetic ramisection, *Proc. Roy Soc Med. (orthop. sect.)* 20 63 1927
- Ryerson, E W Cerebral spastic paralysis in children, *J.A.M.A.* 98 43 1932
- Scaghetti, O The obstetrical shoulder trauma *Surg Gynec. & Obst.* 66 868 1938
- Sever J W Obstetrical paralysis *Am. J Dis. Child.* 12 541 1916
- Sever J W Obstetrical paralysis, *J.A.M.A.* 85 1862 1925
- Sever J W Obstetrical paralysis *Surg Gynec. & Obst.* 44 547 1927
- Steindler A. Nutrition and vitality of the tendon in tendon transplantation, *Am. J Orthop. Surg* 16 63 1918
- Steindler A. Operative treatment of paralytic conditions of the upper extremity *J Orthop Surg.* 1 608 1919
- Steindler, A. *Reconstructive Surgery of the Upper Extremity* New York, Appleton Century 1923
- Steindler, A. Tendon transplantation in the upper extremity *Am. J Surg.* 44 260 1939
- Steindler A. *Orthopaedic Operations*, Springfield, Ill., Thomas 1940 p 315
- Steindler A. Muscle and tendon transplantation at the elbow as American Academy Orthopedic Surgeons *Reconstruction surgery of the extremities*, Ann Arbor Edwards, 1944
- Stoffel, A. The treatment of spastic contracture, *Am. J Orthop. Surg* 10 611 1912 13
- Taylor A. S. Results from surgical treatment of brachial birth palsy, *J.A.M.A.* 48 96 1907
- Thibodeau, A. A., Wagner L. C., and Carr F J, Jr The evaluation of surgical procedures on bones, muscles and peripheral nerves in spastic paralysis *Am J Surg* 43 821 1939
- Thomas T T Laceration of the axillary portion of the capsule of the shoulder joint as a factor in the etiology of traumatic combined paralysis of the upper extremity *Ann. Surg* 53 77 1911
- Thomas, T T Obstetrical or brachial birth palsy *Am. J Obst.* 73 5,7 1916
- Thomas T T Traumatic brachial paralysis with flail shoulder joint *Ann. Surg* 66 532 1917
- Thomas T T Brachial birth palsy a pseudo-paralysis of shoulder joint origin *Am J M. Sc.* 159 207 1920
- Thompson J L. Anatomical methods of approach on operations on the long bones of the extremities *Ann. Surg* 68 309 1918
- Wagoner G Prevention of subluxation of the humeral head following operations for arthrodensis of the shoulder joint, *J Bone & Joint Surg* 15 978 1933
- Watson-Jones R. Extra-articular arthrodensis of the shoulder *J Bone & Joint Surg* 15 862 1933
- Wilson P D Complete rupture of the supraspinatus tendon *J.A.M.A.* 96 433-439 1931

Index

- Abduction of arm 29
 - of humerus 34 35
- Abnormalities congenital *See* Congenital abnormalities
- Abscess epidural 291
- Acromioclavicular joint anatomy normal 16 27 28 195
 - dislocation complete 196-198
 - diagnosis 19, 198
 - roentgenographic examination 198
 - subluxation 195 196 197
 - treatment of old cases 203 204
 - treatment of recent cases complete dislocation 199-203
 - excision of outer end of clavicle 200-201
 - reconstruction of coracoclavicular ligaments 201 203
 - screw transfixion, 203
 - wire transfixion 199-200
 - subluxation 198-199
 - immobilization 199
 - motion 31
 - surgical approach, 390-392
- Acromioclavicular ligament 21
- Acromion process 23
 - anatomy normal 18
 - evolutionary increase 8
 - excision of 417 419
- Acromioplasty 136-137
- Adduction of arm 30
 - as factor in calcareous tendinitis 177
 - in incidence of pathologic changes in musculotendinous cuff 78-79 94
- Amputation(s) 412-417
 - disarticulation 413-416
 - interscapulothoracic 414 415
 - at surgical neck 412 414
- Anesthesia 381
- Angle torsion of humerus 9 10 18-19
- Approach (surgical) to acromioclavicular joint, 390-392
 - anterior 382 386
 - short through deltoid muscle 384
 - anterosuperomedial 384-385
 - anterosuperoposterior 385
 - posterior 388-390
 - of Kocher 388-390
 - of Rowe and Yee 390 391
 - to sternoclavicular joint 390
 - superior 386-388
 - of Henry 388
 - through deltopectoral cleft 382 384
- Approach—(Continued)
 - transacromial 396-397
 - access to interior of joint 397
- Arch coraco-acromial 1, 13 25
- Artery(ies) 373
 - subclavian 286 287
- Arthritis hypertrophic of cervical spine 212 293
 - rheumatoid, of cervical spine 292
- Arthrodesis of scapulohumeral joint 395
 - combined intra and extra articular 401-402
 - by compression 402
 - extra articular 399-400
 - of Puttr 400
 - of Watson Jones 399-400
 - function optimum position of 402-404
 - indications 395
 - intra articular 400-401
 - positions recommended 396-398
 - procedures 399-404
 - and scoliosis 398
- Articular capsule 20-23
 - anatomy normal, 20-23
 - of sternoclavicular joint 2,
- Articulations of shoulder joint 16
 - acromioclavicular 16
 - scapulohumeral 16
 - scapulothoracic 16
 - sternoclavicular 16
- Aspiration and needling for calcareous tendinitis 18, 183
- Axillary nerve course of 377 382
- Axiohumeral muscles evolution of 13
- Axioclavicular muscles evolution of 12
- Biceps brachii muscle 32 35
 - dislocation of long head, 172 175
 - clinical features 174 1 5
 - treatment 175
 - evolution of 13
 - long head, 33
 - short head, 33
 - synovial sheath 21
 - transplantation 406-409
- Biceps tendon 65-69 93 94
 - and bicipital groove 89 91 93
 - evolution of humerus 11
 - lesions and good function 107 109
 - long head 17 19 20 22 23
 - rupture of 139
 - rupture of 169-175
 - clinical features 170-172

Biceps tendon—(Continued)

rupture of—(Continued)

proximal, 169-172

site, 170

spontaneous 169

test Hueter 172

Ludington, 172

Yergason 172

treatment 172

choice of 172

postoperative 172

Bicipital groove 10 11 19 89 91

affected by osseous lesions, 157

altered by fractures, 156

mechanism 90-93

Bicipital tenosynovitis 150-169

association, with cuff tears 120

with trauma 150

case reports 152

clinical features 151

diagnostic tests 151

and frozen shoulder 168

incidence of 150

operative findings 152

pathology of 152

and scalenus anticus syndrome 159

and supraspinatus tears 169

treatment 164-168

conservative 164-166

surgical, 165 168

technic 166-168

without trauma, 161 168

age groups 162 164

case reports 162 163

and frozen shoulder 166

treatment 164-168

Bone cyst, 330

solitary 337 341

clinical features 340

impaired function 340

pain, 340

swelling 340

diagnosis 341

incidence, 338

origin 337

pathology 338-340

microscopic 340

roentgenologic features 340

site 338

treatment of 341

varieties, 337

Bone lesions, metastatic, differential diagnosis 365

pathologic features 365

roentgenographic features, 365

treatment, 365

types, 363-365

Bone tumors 316-366

classification, 316-317

Bone tumors—Continued)

diagnosis, 317 323

age 317

biopsy aspiration, 323

surgical 323

body reaction 320-321

histologic examination 323

history 317 319

injury 318

laboratory examination 322

serum phosphatase, 322 323

loss of function 318

objective findings 319-320

palpation 320

size 320

skin, 319

pain 318

pulsation, 320

rapidity of growth, 317

roentgenographic, 321 322

site of tumor 319

incidence 316

Brachial plexus, anatomy 282 283 286

composition of anterior primary rami 282 283

compression, by hyperadduction of arm, 305

by reduction of costoclavicular space 305

constriction by anomalous structures 303

lesions inflammatory 307

traumatic 308

limb distribution of plexus spinal nerves 282

axis and deep structures 283

of extremity 283

postaxial or ulnar surface 282 283

preaxial or radial surface 282

nerve trunks divisions of 283

formation of 283

in obstetric paralysis, 309

pressure-producing lesions outside vertebral col-

umn, 298

serum neuritis 308

spinal cord branches 283

tumors involving 306

Brachialgia symptom complex related to shoulder

pain, 274

Bursa(e) infraserratus 26

of shoulder joint anatomy normal 25-27

subacromial. See Subacromial bursa

subcoracoid, 25 27

subscapular 26

supra acromial, 26-27

Calcaneous deposits, in supraspinatus tendon, 138

Calcaneous tendinitis 138 177 190

acute syndrome 179 180 182 183

treatment, 184-185

aspiration and needling 187 188

after treatment 187 188

cold packs 190

Calcaneus tendinitis—(Continued)

acute syndrome—(Continued)

treatment—(Continued)

- irrigation 188
- manipulation 189
- roentgen ray therapy 189
- surgical 185-187

age as factor 177

case report 148

chronic syndrome 183 189-190

treatment *See* Calcaneus tendinitis sub-acute and chronic syndrome treatment

etiology constitutional diseases 179

- infection 179
- occupation 178
- trauma 178-179

pathogenesis 179-180

- calcium deposition 180
 - carbon dioxide tension 180
 - mechanism of 180
 - salts 180

"critical zone" 179-180

radiographic characteristics 183-189

localization 184

sex as factor 177

site 177

subacute syndrome 178 181 182 184

subacute and chronic syndrome, calcaneus deposits 189

frozen shoulder treatment 190

treatment 189-190

- cold packs 190
- manipulation 189-190
- physical therapy 189
- roentgen radiation 189

in tendon, supraspinatus 178 179

subscapularis 180

trauma pathology 178-179

radiographic changes, 178

treatment, clinical observations 184

- diathermy 190
- packs (cold and hot) 190
- procaine injection 190

Calcium deposition, 180

- carbon dioxide tension 180
- mechanism of 180

Callus formation massive 255

Capsule, articular *See* Articular capsule synovial 21 23

Carcinoma metastatic, 331 332

- in bone 362 365
- incidence, 362 363

Cartilage, articular of humeral head, 96-99

Cavity glenoid. *See* Glenoid cavity

Cephalic vein, 375

Cervical rib syndrome 298

treatment 306

Cervical spine anatomic considerations 280-285

Cervical spine—(Continued)

cervical nerves 281

disks intervertebral 280-281

foramen intervertebral 281

- deformity in nerve root compression 281
- ligamentous and capsular inflammation causing radiculitis 281

hypertrophic arthritis as cause of radicular pain 281

lordotic curve 281 282

traumatic lesions of vertebrae 293 295

tumors of canal 287

of cord 287

clinical features 283

diagnosis 288

intramedullary 287

radicular pain differential diagnosis 287 288

of spinal nerve roots clinical features 288

of vertebrae multiple 293

primary 293

Chondroblastoma, benign 335 337

Chondroma 356-357

age 356

clinical features 356

incidence 356

malignant transformation of 357

pain, 356

roentgenographic features 356-357

site, 356

swelling 356

treatment 357

Chondrosarcoma 351 356

Clavicle, anatomy normal 20

excision, total 420-421

incision to expose 392

outer end 400

resection inner end, 400

Compression syndromes in supraclavicular region 287

Congenital abnormalities 45 50

of coracoclavicular joint, 50

dislocation of shoulder 50

and obstetric palsy 50

types 50

dysostosis, cleidocranial, 48 49

cleidocranial, characteristics 49

of glenohumeral joint 49 50

concavo-convex, 49 50

glenoid fossa flattened, 49 50

humerus varus 49 50

scapula high, 45-49

operative technic 47-49

Coronoid ligament 21 27

Coraco-acromial ligament 17 18 21 23 25

in bicipital tenosynovitis 168

Coracobrachialis muscle 33

- Biceps tendon—(*Continued*)
 rupture of—(*Continued*)
 proximal, 169-172
 site 170
 spontaneous 169
 test, Hueter 172
 Ludington, 1/2
 Yergason 172
 treatment 172
 choice of 172
 postoperative 172
- Bicipital groove 10 11 19 89-91
 affected by osseous lesions 157
 altered by fractures 156
 mechanism, 90-93
- Bicipital tenosynovitis, 150-169
 association, with cuff tears 120
 with trauma 150
 case reports 152
 clinical features 151
 diagnostic tests 151
 and frozen shoulder 168
 incidence of 150
 operative findings, 152
 pathology of 152
 and scalenus anticus syndrome 159
 and supraspinatus tears, 169
 treatment, 164 168
 conservative 164-166
 surgical 165-168
 technic, 166-168
 without trauma, 161 168
 age groups, 162 164
 case reports 162 163
 and frozen shoulder 166
 treatment, 164-168
- Bone cyst, 330
 solitary 337 341
 clinical features 340
 impaired function, 340
 pain 340
 swelling 340
 diagnosis 341
 incidence 338
 origin, 337
 pathology 338-340
 microscopic 340
 roentgenologic features 340
 site, 338
 treatment of 341
 varieties 337
- Bone lesions metastatic, differential diagnosis 365
 pathologic features, 365
 roentgenographic features 365
 treatment, 365
 types, 363-365
- Bone tumors 316-366
 classification, 316-317
- Bone tumors—(*Continued*)
 diagnosis 317 323
 age, 317
 biopsy aspiration, 323
 surgical 323
 body reaction, 320-321
 histologic examination 323
 history 317 319
 injury 318
 laboratory examination 322
 serum phosphatase 322 323
 loss of function 318
 objective findings 319-320
 palpation, 320
 size 320
 skin 319
 pain, 318
 pulsation 320
 rapidity of growth, 317
 roentgenographic 321 322
 site of tumor 319
 incidence 316
- Brachial plexus anatomy 282 283 286
 composition of anterior primary rami 282 283
 compression by hyperadduction of arm 305
 by reduction of costoclavicular space, 305
 constriction by anomalous structures 303
 lesions inflammatory 307
 traumatic, 308
 limb distribution of plexus spinal nerves 282
 axial and deep structures 283
 of extremity 283
 postaxial or ulnar surface, 282 283
 preaxial or radial surface 282
 nerve trunks divisions of 283
 formation of 283
 in obstetric paralysis 309
 pressure-producing lesions outside vertebral column, 298
 serum neuritis 308
 spinal cord branches 283
 tumors involving 306
- Brachialgia, symptom complex related to shoulder pain, 274
- Bursa(e) infrascapular 26
 of shoulder joint, anatomy normal 25-27
 subacromial. *See* Subacromial bursa
 subcoracoid, 25 27
 subscapular 26
 supra-acromial, 26-27
- Calcareous deposits in supraspinatus tendon, 138
 Calcareous tendinitis 138 177 190
 acute syndrome 179 180 182 183
 treatment, 184-185
 aspiration and needling 187 188
 after treatment 187 188
 cold packs 190

Fractures of anatomic neck of humerus — (Continued)

- impacted 254
 - of anatomic head into greater tuberosity 255-256
 - collar-cuff ring 254
 - exercise 254
 - prognosis 254
 - mechanism 244-252

- adduction and abduction type 250
- comminuted fracture 251-252
- direct force 249
- factors affecting type of lesion 250-252
- indirect force 249
 - false dislocation 241
 - true dislocation 250
 - in old age 250

neck, anatomic. See Fractures of anatomic neck of humerus

- comminuted type 260
- and head. See Fractures of head and neck of humerus
- occurrence 260
- surgical. See Fracture of surgical neck of humerus

- treatment of impacted type 260
 - indications for 260

- requiring no reduction 252-254

- requiring reduction 256-264

- fracture of greater tuberosity with displacement 256-259. See also Fracture of greater tuberosity

- indications 252

- method of treatment 252

- tuberosity greater. See Fractures of greater tuberosity

- lesser. See Fractures of lesser tuberosity

- unimpacted 254
- of lesser tuberosity with retraction occurrence 259

- pathology 251

- postoperative management 260

- treatment 260

- of surgical neck of humerus with complete displacement 260-262

- abduction apparatus 263

- deformity 260

- treatment by manipulative reduction 260-262

- Fracture-dislocation of greater tuberosity 268-270

- associated cuff damage 268

- evaluation of cuff damage and fragment position 268

- occurrence 268

- reduction and management 268

- surgical repair 268-270

- torso cuff 270

Fracture dislocation—(Continued) of humerus types 264

- frozen shoulder 24-135-135-135
- with calcareous tendinitis treatment 135
- case report 134

- characteristics of 130

- etiology 143

- exercise 146

- fixation 134

- after immobilization 144

- manipulation 14

- pathogenesis 145

- pathology 144

- resolution of 145

- roentgenograms 141

- symptoms 141

- treatment 146

Giant-cell tumor 323-334-422

- clinical features 32-325

- joint 32-325

- diagnosis 330-332

- epiphyseal chondromata benign chondroblastoma treatment 336

- clinical features 335

- diagnosis 336

- microscopic features 336

- origin 335

- prognosis 337

- roentgen features 335-336

- etiology 324-325

- incidence 324

- malignant 330-331

- pathology macroscopic 325

- microscopic 325-327

- stromal pattern 325-327

- physical findings 328

- prognosis 334

- roentgenologic findings 328-330

- site 324

- treatment 332-334

- amputations 334

- curettage 333

- irradiation 334

- resection 334

- Gill combined intra and extra articular arthrodesis 401-402

Girdle pectoral. See Pectoral girdle

Glenohumeral joint bursal side 80-94

- alterations degeneration 93

- biceps tendon 93-94

- bicipital groove 90-93

- mechanism 90-93

- bursa subacromial 80-81

- floor 81-83

- rotator cuff 84-90

- complete tears 86

- Coracoclavicular joint, congenital abnormalities 50
 deformities 50
- Coracoclavicular ligament 20 27
 evolution of in mammals, 5
- Coracohumeral ligament 17 23 24
 in bicipital tenosynovitis 168
- Coracoid ligament evolution of in mammals 5
- Coracoid process 17 20 23
 anatomy normal 18
 evolutionary increase 8
- Costoclavicular ligament 21
- Costoclavicular syndrome 304 305
- Costocoracoid membrane 319
- Cuff musculotendinous *See* Musculotendinous cuff
 rotator *See* Rotator cuff
- Cyst of bone. *See* Bone cyst
- Deltoid muscle, 32 34 35 37
 anatomy surgical 314
 insertion on shaft of humerus evolution of 11
 paralysis of 41-44
 muscle transplants 404
 tendon transplant for 409
 transplantation of part 408-410
- Depressed fracture of greater tuberosity complications 255
 mechanism 254-255
 pathogenesis 255
 treatment 255
- Depression, of humerus 35 36
- Dermatomes, 275-277
- Disarticulation of shoulder 413-416
- Disk, articular of sternoclavicular joint, 27
 fibrocartilaginous of sternoclavicular joint, 27
 intervertebral, of cervical region, protrusion, 295-297
- Dislocation of shoulder congenital, 50
 types 50
 and obstetric palsy 50
- Dislocation of shoulder joint, 192 245
- Dysostosis cleidocranial 48 49
 characteristics 49
- Elevation, of arm, 28-29
- Epiphyseal chondromatous giant-cell tumor 331 335-337
- Epiphysis of humerus *See* Humeral epiphysis
- Erb-Duchenne paralysis, 311
- Ewing's tumor 364-365
- Excision of humeral head, Jones technic, 212 213
 reconstruction of musculotendinous cuff 272
- Fascial transplants for paralysis of levator muscles of scapula, 410
 of levator scapulae muscles, 411-412
- Fascial transplants—(*Continued*)
 of rhomboid muscles 411-412
 of serratus anterior muscle 410-411
 about shoulder girdle, 409-410
 of spinal muscles 410
- Fibrosarcoma of bone 344-346
 medullary 346
 prognosis in, 346
- Flexion dorsal, of arm 30
 forward, of arm 29
 of humerus 34-35
- Fossa glenoid, 19 22
- Fracture(s) of anatomic neck of humerus, 263-264
 combined lesions, 263
 occurrence 263
 treatment, 263 264
 depressed, of greater tuberosity 254-255
 and fracture-dislocations of humerus 248-273
 bony union, 248-249
 glenohumeral joint motion 248
 gliding mechanisms, 249
 rotator cuff and fracture fragments, 249
 stability of the glenohumeral joint, 248
 treatment objectives 248
 of greater tuberosity depressed. *See* Depressed fracture of greater tuberosity
 with displacement under acromion 256
 pathology 256-257
 postoperative management, 258
 rupture of musculotendinous cuff 256
 sequelae, 257
 treatment, 251 258
 downward and outward, 258-259
 treatment 259
 and glenohumeral dislocation 256
 retraction of tuberosity downward and outward, 258
 of head and neck of humerus, with dislocation, 270-273
 occurrence, 210
 pathology 210-271
 treatment, reduction 271 272
 transacromial approach of McLaughlin, 271
 of humerus classification anatomic, 252
 therapeutic 252
 depressed fracture of greater tuberosity *See* Depressed fracture of greater tuberosity
 factors affecting type of lesion, direction of arm in fall 250
 falling body 250
 rotation of humeral shaft 250
 head, impacted, into greater tuberosity 255 256
 and neck. *See* Fractures of head and neck of humerus

- Humerus—(Continued)**
 neck anatomy 19
 o tomy of for ob tetric paralysis 313
 tuberosity greater 1, 19
 lesser 19
 upper end, resection 421-422
 varus 49 50
- Herpes zoster** 290-291
- Hypertrophic osteodystrophy**, 331
- Impacted fractures of anatomic head into greater tuberosity** 255 256
- Incision** See also Approach (surgical)
 anterosuperoposterior 385-386
 bursal 383
 deltopectoral 382
 to expose clavicle 392
 to expose scapula 392 395
 saber-cut 38, 383
 of skin 381 382
 transaxillary 380
- Index, infraspinatus** 8
 scapular progressive decrease 1
- Infraspinatus muscle** 17 19 23 24 35 38
- Innervation** sympathetic, of upper extremity 285
- Interclavicular ligament** 2
- Intertubercular groove** 89 91
- Intervertebral disk** physiologic function, 280
 effect on traumatized nerve roots 280
 role in mechanism of radicular pain 280
- Joint acromioclavicular anatomy** normal, 16 27 28
 dislocation 195 204
 motion, 31
 surgical approach, 390-392
 glenohumeral. See Glenohumeral joint
 scapulohumeral. See Scapulohumeral joint
 scapulothoracic 16
 shoulder See Shoulder joint
 sternoclavicular anatomy normal 16 26 27
 dislocations 192 195
 motion, 31
 surgical approach, 390
- Junction epiphyseal**, 20
- Kieppel Feil deformity** 298
- Kleinberg's operation**, for deformities of humerus 314
- Klumpke paralysis** 311
- Kocher incision**, to expose entire scapula, 393
- Labrum glenoidale** 19 20 22 61 93
 anatomy normal, 18
- Landmarks bony in surgery** 375
 of shoulder girdle 374
- Latissimus dorsi muscle** 33 37
- Lesions and good function** 94-110
 articular cartilage of humeral head 106 107
 biceps tendon 10, 109
 clinical cases 95 96
 natural reformation 110
 size 103-109
 ulcus 100
 synovial membrane and musculotendinous cuff 100-107
 osteoblastic 364 365
 osteolytic 364 365
- Levator muscles of scapula** paralysis fascial transplants 410
- Levator scapulae muscle** 36 38
 paralysis fascial transplants 411-412
- Ligament acromioclavicular** 21
 coroid, 21 27
 coraco-acromial 1, 13 21 23-25
 in bicipital tenosynovitis 163
 coracoclavicular 20 27
 evolution of in mammals 5
 coracohumeral 1, 23 24
 in bicipital tenosynovitis 163
 coracoid, evolution of in mammals 5
 costoclavicular 2,
 glenohumeral. See Glenohumeral ligament
 humeral transverse 17 21
 interclavicular 2,
 rhomboid, 27
 sternoclavicular 2,
 suprascapular 17 20
 suspensory for humeral head, 23 24
- Limb pectoral** See Pectoral limb
- Ludington test** 1, 2
- Luxatio erecta** 207 224
- Luxation of cervical spine "creeping migration"** of vertebrae 293-295
 shoulder and arm pain mechanism, 293 295
- Manometric study** as diagnostic aid, 288
- Mechanical force requirements of shoulder joint** 32 38
- Membrane costocoracoid**, 319
 synovial, 22 69-73 75
 hypertrophy 10 72
 lesions, musculotendinous cuff 100-107
 pathology incidence 10-73
 site 10
- Motion at shoulder joint** 30-32
 acromioclavicular 31
 glenohumeral 30-31
 muscle force couple 30
 sternoclavicular 31
- Movements at shoulder joint** 28 30
 abduction, 29 34-35
 adduction 30
 depression 35-36
 elevation 28-29

Glenohumeral joint—(Continued)

- rotator cuff—(Continued)
 - iraying 85-86
 - lesions large 87 90
 - small, 86-87
 - shredding, 85 86
 - tuberosities, 90-93
 - variations 94
- congenital abnormalities 49 50
- concavo-convex 49 50
- glenoid fossa flattened, 49 50
- dislocation 211
- dislocation (recurrent) 229-244
 - bone changes 235
 - fibrous capsule 232 234
 - labrum glenoidale, 232 234
 - musculotendinous cuff 232
 - operative treatment, 236-244
 - Bankart, 237 239-242
 - block bone operation 237
 - Henderson 237 242 243
 - postoperative, 243
 - Magnuson, 238-239
 - analysis 244
 - postoperative 239
 - Nicola, 237 243-244
 - postoperative 244
 - plastic procedures on capsule and tendons, 237 238
 - Putti Platt 237 238 241 242 243
 - suspension 236-237
- pathogenesis, 236
- pathology 230-235
- subacromial recesses, 233 234
- glenoid side 53-80
 - alteration in articular cartilage 60-61
 - macroscopic 56-58
 - microscopic, 58-59
 - in subchondral bone 60-61
 - anatomy 53 55
 - biceps tendon, 65 66-69
 - cases 53
 - glenohumeral ligament, 73-77
 - inferior 77
 - middle 74-76
 - superior 6-77
 - variations, 77
 - glenoid cavity 55-62 65 72
 - labrum glenoidale 61 93
 - musculotendinous cuff 77 80
 - anatomy 77
 - lesions 8 94
 - pathology age incidence 78-79 94
 - origin of lesion, 61-62
 - synovial membrane 69-73 75
 - hypertrophy 70 72
 - pathology site, 70
 - incidence, 70-73

Glenohumeral joint—(Continued)

- glenoid side—(Continued)
 - synovial recess 73-75
 - motion 30-31
- Glenohumeral ligament 73-77
 - inferior 19 22 77
 - middle 19 22 74-76
 - relationships 76
 - superior 19 76-77
 - variations 77
 - types, 75
- Glenoid cavity 55-62 65 72
 - anatomy normal, 18 19
 - labrum glenoidale, 18
- Glenoid fossa flattened, 49 50
- Gliding mechanisms of shoulder muscles 3/3
- Groove bicipital. *See* Bicipital groove
- Intertubercular. *See* Intertubercular groove
- Guillain Barré syndrome 291
- Hueter test 1/2
- Humeral epiphysis separation of 264-268
 - displacement 264
 - growth disturbances 265
 - mechanism 264
 - pathology 265
 - sequelae 268
 - treatment 265 268
 - displacement, complete, 267
 - moderate 266-267
 - open reduction 267 268
 - slight, 265-266
- Humeral ligament, transverse 17 21
- Humerus angle torsion, 18-19
 - anatomy normal, 18-20
 - deformities of Kleinberg's operation 314
 - deltoid insertion, evolution of 11
 - epiphysis. *See* Humeral epiphysis
 - evolution of biceps tendon 11
 - bicipital groove 10 11
 - pectoral limb 9
 - alterations in, 9
 - torsion angle, 10
 - fracture-dislocations. *See* Fracture-dislocation, of humerus
 - fractures. *See* Fractures, of humerus
 - groove bicipital. *See* Bicipital groove
 - head, anatomic 19
 - impacted fractures into greater tuberosity 255-256
 - functional disability 2 2
 - ossification 44-45
 - resection, 422-423
 - surgical excision of 272 2 3
 - muscles abductors 34-35
 - depressors 35-36
 - flexors 34-35

- Humerus—(Continued)**
 neck anatomic 19
 osteotomy of for obstetric paralysis 313
 tuberosity greater 1, 19
 lesser 19
 upper end, resection 421-422
 varus 49, 50
- Herpes zoster** 290-291
- Hyperparathyroidism** 331
- Impacted fractures of anatomic head into greater tuberosity** 255, 256
- Incision** *See also* Approach (surgical)
 anterosuperoposterior 385, 386
 bursal 383
 deltopectoral 382
 to expose clavicle 392
 to expose scapula 392, 393
 saher-cut 381, 383
 of skin 381, 382
 transacromial 380
- Index** infraspinatus 5
 scapular progressive decrease 7
- Infraspinatus muscle** 17, 19, 23, 24, 35, 34
- Innervation** sympathetic of upper extremity 265
- Interclavicular ligament** 2
- Intertubercular groove** 39, 91
- Intervertebral disk** physiologic function 240
 effect on traumatized nerve roots 240
 role in mechanism of radicular pain 250
- Joint** acromioclavicular anatomy normal 16, 2
 28
 dislocation 195, 204
 motion 31
 surgical approach 390-392
 glenohumeral *See* Glenohumeral joint
 scapulohumeral *See* Scapulohumeral joint
 scapulothoracic 16
 shoulder *See* Shoulder joint
 sternoclavicular anatomy normal 16, 26, 27
 dislocations 192, 195
 motion 31
 surgical approach 390
- Junction** epiphyseal 20
- Kieppel Feil deformity** 298
- Kleinberg's operation** for deformities of humerus 314
- Kumpke paralysis** 311
- Kocher incision** to expose entire scapula 393
- Labrum** glenoidale 19, 20, 22, 61, 93
 anatomy normal 18
- Landmarks** bony in surgery 375
 of shoulder girdle 374
- Latissimus dorsi muscle** 33, 37
- Lesions and good function** 94, 110
 articular cartilage of humeral head 94-95
 biceps tendon 101, 109
 clinical cases 95, 97
 natural restoration 110
 size 105-109
 ulcus 109
 synovial membrane and musculotendinous cuff 100-101
 osteolytic 364, 365
 osteotic 364, 365
- Levator muscles of scapula** paralysis facial transplants 410
- Levator capulae muscle** 36, 33
 paralysis facial transplants 411-412
- Ligament** acromioclavicular 21
 coronoid 21, 22
 coracoacromial 17, 18, 21, 23, 25
 in bicipital tenosynovitis 164
 coracoclavicular 20, 22
 evolution of in mammals 3
 coracohumeral 17, 23, 24
 in bicipital tenosynovitis 163
 coracoid evolution of in mammals 3
 costoclavicular 2
 glenohumeral *See* Glenohumeral ligament
 humeral transverse 17, 21
 interclavicular 2
 rhomboid 2
 sternoclavicular 2
 supra capular 17, 20
 suspensory for humeral head 23, 24
- Limb** pectoral *See* Pectoral limb
- Ludington test** 1, 2
- Luxatio erecta** 20, 224
- Luxation of cervical spine** "creeping migration" of vertebrae 293, 295
 shoulder and arm pain mechanism, 293, 295
- Manometric study** as diagnostic aid, 285
- Mechanical force requirements of shoulder joint** 32, 33
- Membrane** costocoracoid 3, 9
 synovial 22, 69-73, 75
 hypertrophy 0, 12
 lesions musculotendinous cuff 100-101
 pathology incidence 0-73
 site 10
- Motion at shoulder joint** 30-32
 acromioclavicular 31
 glenohumeral 30-31
 muscle force couple 30
 sternoclavicular 31
- Movements at shoulder joint** 28, 30
 abduction 29, 34, 35
 adduction 30
 depression 35, 36
 elevation 28-29

Movements at shoulder joint—(Continued)

- flexion, 34-35
 - dorsal, 30
 - forward, 29
- position, pivotal, 29
- rotation of scapula, 36-38
- rhythm, scapulohumeral, 30
- Multiple myeloma 360-362
 - clinical features 360-362
 - pain, 360
 - reactions, 361
 - differential diagnosis 360
 - incidence, 360
 - pathology 362
 - prognosis, 362
 - roentgenographic features 362
 - treatment of 362
 - varieties of 360
- Muscle, axohumeral, evolution of 13
- axoscapular group evolution of 12
- biceps brachii *See* Biceps brachii muscle
- coracobrachialis 33
- deltoid. *See* Deltoid muscle
- evolution of pectoral limb 11
- formation from embryonic buds 3
- of humerus abductors 34-35
- depressors 35-36
- flexors, 34-35
- infraspinatus 17 19 23 24 35-38
- latissimus dorsi, 33 37
- levator scapulae, 36 38
 - paralysis, fascial transplants, 411-412
- pectoralis major 32 34 35 375
 - minor 32 33 36 286
- rhomboides major 37 38
 - minor 38
 - paralysis fascial transplants 411-412
- rotator short 374
- scalenus anticus 285-287
 - medius, 285 286
- of scapula, rotators 36-38
- scapulohumeral group, evolution of 11
- serratus anterior 32 35 37
 - paralysis 41-43
 - fascial transplant 410-411
- sternocleidomastoid, 32
- subclavius, 36
- subscapulars 19 21 23 24 33 35 36
- supraspinatus 17 19 23 24 33 36 38
 - function 114
- teres major 33 38
 - minor 19 23 24 35 38
- transplantations, 404-409
- trapezius, 32 34-37
 - paralysis of 42-44
 - transplantation of fascial extension, 405-407
- triceps brachii evolution of 13
- transplantation, 406-409

- Muscle force couple 30 33-34
 - impairment of with isolated nerve paralysis and other lesions 40-44
 - principle of 38-39
 - in scapular rotation, 34
- Musculocutaneous nerve 378
- Musculotendinous cuff 19 24 376
 - abduction of arm role of rotator muscles 120-121
 - anatomy 77 119
 - calcareous deposits pathogenesis 180-183
 - acute syndrome, 182 183
 - chronic syndrome, 183
 - clinical features, 181
 - subacute syndrome 181 182
 - symptoms 181
 - and glenohumeral joint, 77 80
 - inadequacy of 114
 - lesions 78 94
 - degenerative 99
 - and synovial membrane, 100-107
 - massive avulsion from shock therapy 119
 - minor damage, painful arc of motion 120
 - pathologic alterations 180-183
 - prime function, 114
 - ruptures of *See* Ruptures of musculotendinous cuff
 - tendinitis calcareous *See* Calcareous tendinitis
- Myelography as diagnostic aid, 288 297
- Myeloma, multiple. *See* Multiple myeloma
- of cervical vertebrae, 293
- plasma-cell, solitary 362
 - differential diagnosis 362
 - prognosis, 362
- Myelo-radiculo-neuritis *See* Guillain-Barré syndrome
- Mycosis ossificans circumscripta 365 366
 - clinical features 366
 - incidence 366
 - malignant degeneration 365
 - roentgenographic features 366
 - sites 366
 - treatment, 366
- Neoplasm *See* Tumor
- Nerve(s) anatomy surgical, 376
 - axillary course of 377 382
 - musculocutaneous, 378
 - supply segmental, skeletal muscles of neck, shoulder and upper arm, 284 285
 - suprascapular 378
- Neuroma 307
- Neuroblastoma 364-365
- Occupation as factor in calcareous tendinitis, 178
- in rupture of musculotendinous cuff 114 119

- Osteoblastic lesions 364 365
 Osteochondroma 35, 360
 age 358
 clinical features 358-359
 local paralysis 359
 etiology 35, 358
 familial tendency 357
 malignant transformation 354
 quiescent lesions 358
 roentgenographic features 359
 site 358
 treatment 359-360
 Osteoid osteoma 349-351
 clinical features 350
 differential diagnosis of 349
 incidence of 349
 pathology 351
 roentgenologic features 350-351
 site of 349
 spontaneous arrest 351
 treatment 351
 block reaction 351
 Osteolytic lesions 364 365
- Pachymeningitis, cervical, hypertrophic 289-290
 Paget's osteitis deformans malignant transformation 354
 Pancoast tumor 306 307
 Paralysis, of deltoid muscle, 40-41
 transplants for 404
 lower arm 311
 of nerves with impairment of muscle force couple 40-44
 obstetric 308
 clinical features 308
 and congenital dislocation of shoulder 50
 etiology and pathogenesis 309
 osteotomy of humerus 313
 postoperative management 313
 Sever's operation 312
 treatment 310
 surgical procedures 312
 types of 311
 of serratus anterior muscle 41-43
 of trapezius muscle 42-44
 upper arm 311
 whole arm 311
 Pectoral girdle evolution of 2
 amphibia 3
 birds 3
 fishes 2 4 5
 reptiles 3
 mammals 5
 ligament coracoid, 5
 coracoclavicular 5
 phylogenesis 6
- Pectoral limb 1
 anatomy comparative 1
 evolution of in extremities upper 5
 muscles 11
 humerus 9
 axiohumeral group 13
 axioscapular group 12
 biceps brachii 13
 biceps tendon 11
 bicipital groove 10
 morphologic alterations in 9
 scapulohumeral group 11
 torsion angle 10
 triceps brachii 13
 scapula 6
 infraspinous index 3
 morphologic changes in scapular index 6 8
 origin 1
 Pectoralis muscle major 32 34 35 3,5
 minor 32 33 36 256
 Periarthritis scapulohumeral 133
 Platybasia (basilar compression) 290
 Plexus brachial. See Brachial plexus
 Position of patient for surgery 350
 pivotal of arm 29
 Process acromion 23
 anatomy normal 18
 evolutionary increase 3
 excision of 41"-419
 coracoid, 17 20 23
 anatomy normal 18
 evolutionary increase 3
 Protrusion of cervical intervertebral disk 295-297
 anterior root involvement 297
 differential diagnosis 297
 localization of lesions 296
 motor phenomena 296
 myelography 297
 occurrence 295
 pain variations 296
 radiographic findings 297
 symptoms 295
 trauma as factor 295
 treatment 297
 Putti extra articular arthrodesis 400
- Queckenstedt test 288
- Recess infraspinatus 19
 subscapularis, 19 21 22
 synovial 73-74
 Referred pain cutaneous distribution of roots in the extremities, 279
 in the trunk, 279
 dermatome areas in localization 276 277
 effect of stretching on nerve root 280

Movements at shoulder joint—(Continued)

- flexion, 34-35
 - dorsal, 30
 - forward, 29
- position, pivotal, 29
- rotation of scapula, 36-38
- rhythm, scapulohumeral, 30
- Multiple myeloma 360-362
 - clinical features 360-362
 - pain, 360
 - reactions 361
 - differential diagnosis 360
 - incidence 360
 - pathology 362
 - prognosis, 362
 - roentgenographic features 362
 - treatment of 362
 - varieties of 360
- Muscle axiohumeral, evolution of 13
- axioscapular group evolution of 12
- biceps brachii. *See* Biceps brachii muscle
- coracobrachialis, 33
- deltoid. *See* Deltoid muscle
- evolution of pectoral limb 11
- formation from embryonic buds 3
- of humerus abductors 34-35
 - depressors, 35-36
 - flexors, 34-35
- infraspinatus 17 19 23 24 35-38
- latissimus dorsi, 33 37
- levator scapulae, 36 38
 - paralysis fascial transplants 411-412
- pectoralis major 32 34 35 35/5
 - minor 32 33 36 286
- rhomboides major 37 38
 - minor 38
 - paralysis fascial transplants, 411-412
- rotator short, 374
- scalenus anticus, 285 287
 - medius, 285 286
- of scapula, rotators, 36-38
- scapulohumeral group evolution of 11
- serratus anterior 32 35-37
 - paralysis 41-43
 - fascial transplant, 410-411
- sternocleidomastoid, 32
- subclavius, 36
- subscapulars, 19 21 23 24 33 35 36
- supraspinatus 17 19 23 24 33 36 38
 - function, 114
- teres major 33 38
- minor 19 23 24 35-38
- transplantations, 404-409
- trapezius, 32 34-37
 - paralysis of 42-44
 - transplantation of fascial extension, 405-407
- triceps brachii, evolution of 13
- transplantation, 406-409

- Muscle force couple 30 33-34
 - impairment of with isolated nerve paralysis and other lesions, 40-44
 - principle of 38-39
 - in scapular rotation, 34
- Musculocutaneous nerve 378
- Musculotendinous cuff 19 24 376
 - abduction of arm role of rotator muscles 120-121
 - anatomy 77 119
 - calcareous deposits pathogenesis 180-183
 - acute syndrome, 182 183
 - chronic syndrome 183
 - clinical features, 181
 - subacute syndrome, 181 182
 - symptoms 181
 - and glenohumeral joint 77-80
 - inadequacy of 114
 - lesions, 78 94
 - degenerative, 99
 - and synovial membrane 100-107
 - massive avulsion from shock therapy 119
 - minor damage, painful arc of motion 120
 - pathologic alterations 180-183
 - prime function, 114
 - ruptures of. *See* Ruptures of musculotendinous cuff
 - tendinitis calcareous. *See* Calcareous tendinitis
- Myelography as diagnostic aid, 288 297
- Myeloma, multiple. *See* Multiple myeloma
- of cervical vertebrae 293
- plasma-cell, solitary 362
 - differential diagnosis 362
 - prognosis, 362
- Myelo-radiculo-neuritis. *See* Guillain-Barré syndrome
- Myositis ossificans circumscripta, 365-366
 - clinical features, 366
 - incidence 366
 - malignant degeneration, 365
 - roentgenographic features, 366
 - sites, 366
 - treatment 366
- Neoplasm. *See* Tumor
- Nerve(s) anatomy surgical, 376
 - axillary course of 377 382
 - musculocutaneous 378
 - supply segmental, skeletal muscles of neck, shoulder and upper arm 284 285
 - suprascapular 378
- Neurinoma 307
- Neuroblastoma, 364-365
- Occupation as factor in calcareous tendinitis 1/8
 - in rupture of musculotendinous cuff 114 119

- Osteoblastic lesions 364 365
 Osteochondroma 357 360
 age 358
 clinical features 358-359
 local paralysis 359
 etiology 357 358
 familial tendency 357
 malignant transformation 354
 quiescent lesions 358
 roentgenographic features 359
 site 358
 treatment 359-360
 Osteoid osteoma 349-351
 clinical features 350
 differential diagnosis of 349
 incidence of 349
 pathology 351
 roentgenologic features, 350-351
 site of 349
 spontaneous arrest 351
 treatment 351
 block reaction, 351
 Osteolytic lesions, 364 365
- Pachymeningitis cervical hypertrophic 289-290
 Paget's osteitis deformans malignant transformation, 354
 Pancoast tumor 306 307
 Paralysis of deltoid muscle 40-41
 transplants for 404
 lower arm 311
 of nerves, with impairment of muscle force couple 40-44
 obstetric 308
 clinical features, 308
 and congenital dislocation of shoulder 50
 etiology and pathogenesis 309
 osteotomy of humerus 313
 postoperative management 313
 Sever's operation 312
 treatment 310
 surgical procedures 312
 types of 311
 of serratus anterior muscle, 41-43
 of trapezius muscle 42-44
 upper arm 311
 whole arm 311
 Pectoral girdle evolution of 2
 amphibia 3
 birds 3
 fishes 2 4 5
 reptiles 3
 mammals, 5
 ligament coracoid, 5
 coracoclavicular 5
 phylogenesis 6
- Pectoral limb 1
 anatomy, comparative 1
 evolution of in extremities upper 5
 muscles 11
 humerus 9
 axiohumeral group 13
 axioscapular group 12
 biceps brachii 13
 biceps tendon 11
 bicipital groove 10
 morphologic alterations in 9
 scapulohumeral group 11
 torsion angle 10
 triceps brachii 13
 scapula, 6
 infraspinous index 8
 morphologic changes in scapular index 6 8
 origin 1
 Pectoralis muscle major 32 34 35 375
 minor 32 33 36 286
 Periarthritis scapulohumeral 138
 Platybasia (basilar compression) 290
 Plexus brachial *See* Brachial plexus
 Position of patient for surgery 380
 pivotal of arm 29
 Process, acromion 23
 anatomy normal, 18
 evolutionary increase 8
 excision of 417-419
 coracoid, 17 20 23
 anatomy normal, 18
 evolutionary increase 8
 Protrusion of cervical intervertebral disk, 295-297
 anterior root involvement 297
 differential diagnosis, 297
 localization of lesions, 296
 motor phenomena, 296
 myelography 297
 occurrence, 295
 pain variations 296
 radiographic findings 297
 symptoms 295
 trauma as factor 295
 treatment 297
 Putti, extra articular arthrodesis, 400
- Queckenstedt test 288
- Recess infraspinatus, 19
 subscapularis 19 21 22
 synovial 73 74
 Referred pain cutaneous distribution of roots in
 the extremities 279
 in the trunk 279
 dermatome areas in localization, 276 277
 effect of stretching on nerve root 280

Referred pain—(Continued)

- incomplete involvement of the nerve root, theory of Eaton 279
- maneuvers of spine causing nerve root traction 280
- role of epidural veins in nerve root compression, 279
- segmental pattern, investigation by Kellgren and Lewis 275-278
 - in radicular or root pain, 278
- skin and deep pain distinguishing features 278

Resection(s) 411-423

- acromion process, excision 417-419
- of clavicle, excision total, 420-421
 - inner end 400
 - outer end, 420
- of humerus, head, 422-423
 - upper end, 421-4 2
- of scapula, 419-420

Rhomboid ligament, 27

- Rhomboides muscle major 37 38
- minor 38

paralysis fascial transplants 411-412

Rhythm, scapulohumeral 30

Rib cervical, bilateral, 401

- syndrome 298
- treatment 306
- vascular phenomena 300

Rim rents 116

Rotation of scapula, 36-38

Rotator cuff 84-90

- complete tears 86
- fraying 85-86
- lesions, large 87 90
- small 86-87
- shredding 85 86

Rotator muscles about 3 4

Rupture(s) See also Tears

- of biceps tendon, long head, 139
- of supraspinatus tendon, 41 139

Ruptures of musculotendinous cuff 41 112 137

- areas involved, 113
- complete 113-114
 - atrophy of rotator muscles 123-125
 - clinical features, age, 119
 - history 119
 - local, 122 127
 - pain 119-120
 - function, impairment of 120-122
 - hypertrophy of deltoid, 125
 - internal derangement of shoulder 123
 - jog and soft crepitus, 123
 - occupation, 119
 - massive avulsion treatment, surgical, Nicola procedure, 134
 - radiographic examination, 125
 - recent treatment, conservative, 128

Ruptures of musculotendinous cuff—(Continued)
complete—(Continued)

- synovial fluid, 125
- tears eminence and sulcus 126
- tenderness, at deltoid insertion, 123
 - local, 122 123
- tension pain, 120
- thickened bursal walls 125
- treatment, surgical, 128 131 137
 - Bosworth method, 132
 - Codman method, 131 132
 - division of coraco-acromial ligament, 133
 - excision of bursa, 133
 - Jones method, 132
 - McLaughlin method, 132 133
 - massive avulsion, 134
 - postoperative management, 133 134
 - Wilson method, 132
- concomitant lesions 134-136
 - bursa, excision of 136
 - bursitis subacromial 136
 - calcareous deposits, 135
 - dislocations and fractures, 136
 - exostoses, 136
 - tendinitis 135
 - frozen shoulder 135
 - tenosynovitis bicipital, 134-135
- frequency of 113
- and good function 105 107
- lesions of synovial side 113
- massive avulsions recession of tuberosities 125
- mechanism direct injuries 115
- indirect strains 115 116
- supraspinatus region attrition 113
 - complete 116-127
 - clinical features 119-127
 - longitudinal or vertical rents 117
 - massive avulsion, 118-119
 - retraction of 117 118
 - tract of greater tuberosity 117
 - transverse 116-117
 - mechanical and anatomic 113-114
 - occupations 114
 - senescence 113
- tears, surgical treatment acromioplasty anteroposterior osteotomy 137
- treatment surgical, 128-137
 - acromioplasty oblique osteotomy 137
 - postoperative management exercises 134

Sarcoma of bone Codman's triangle 342

- dissemination of 342
- gradation of 341 342
- incidence 341
- pathologic fracture in, 342
- site 341

Sarcoma of bone—(Continued)

- chondroblastic primary 351 354
 - age incidence 351
 - metastasis 352
 - pain 351
 - pathology 352
 - prognosis 354
 - roentgenographic features 352
 - treatment 353-354
 - amputation 354
- secondary 356
 - incidence 354
 - pathology 354
 - recurrences, 354-356
 - roentgenographic features 353 355
 - site, 354
 - treatment 356

Ewing's, 346-349

- clinical features 34, 348
- histology 348
- incidence 346
- origin, 346
- pathology 348
- prognosis, 349
- treatment 348-349
- osteogenic, 331 342
 - medullary 344
 - osteolytic, 343-344
 - roentgenology 344
- sclerosing, clinical course 342
- physical signs 342

Scalenus anticus muscle, anatomy 285 287

Scalenus anticus syndrome 301 305

and bicipital tenosynovitis 159

operation for 300

symptoms and signs 305

types 286

Scalenus medius muscle anatomy 285-286

Scapula anatomy normal, 17 18

blade of 18

elevated, 45-49

evolution of pectoral limb 6

infrascapular index, 8

morphologic changes in scapular index 6 8

high 45-49

incisions to expose 392 395

muscles 36-38

resection 419-420

rotation of muscle force couple 34

spine of 17 18 20

evolutionary increase 8

undescended, 45-49

Scapulohumeral joint, acromion process *See*

Acromion process

anatomy normal, 16-23

arch, coraco-acromial, 17 18 25

Scapulohumeral joint—(Continued)

arthrodesis 395

articular capsule 20-23 27

clavicle *See* Clavicle

coracoid process 8 1, 18 20 23

deformities 49 50

dislocation 204 223

acute 204 205

incidence 204

clinical features 214 223

examination 215 216

history 215

pain 215

palpation 215

mechanism 206 20, 209

falls 207 209

hyperabduction 209

hyperextension 209

nerve injuries axillary circumflex, 212 213

musculocutaneous 213 214

pathology 209-212

position humeral head and capsule 209-212

humeral head and capsule head inside 210-211

humeral head and capsule head outside, 209 210

humeral head and glenoid cavity 209

treatment 216-223

anesthesia for 216

clinical facts governing 221 223

Hippocrates maneuver 219-220

Kocher's maneuver 216-219

Nicola's maneuver 220 222

post reduction, 220-221

traction on adducted humerus 216 217

external rotation 219

forward adduction, 219

internal rotation, 219

traction and leverage with foot in the axilla 219-220

types, 205 207

luxatio erecta, 207 224

posterior 205

subclavicular 205

subcoracoid 205 207

subglenoid, 205 207

subspinous 205

vascular injuries 214

glenoid cavity *See* Glenoid cavityhumerus. *See* Humerusscapula *See* Scapula

Scapulohumeral muscles evolution of 11

Scapulohumeral periarthritis 138

Scapulohumeral rhythm in cuff tears 121 122

normal relation of scapular to humeral motion 121 122

- Scapulothoracic joint, anatomy normal, 16
- Sclerotomes 277
- Scoliosis, and arthrodesis of scapulohumeral joint, 398
- Segmental pain deep observation by Inman and Saunders 277
- false localization, 278
- Segmental reference of pain to shoulder and arm causative factors 275
- neurogenic lesions objective manifestations 275
- Senescence and ruptures of musculotendinous cuff 113
- Serratus anterior muscle 32 35 37
- paralysis, 41-43
- fascial transplant, 410-411
- Serum neuritis, of brachial plexus 308
- Sever's operation, for obstetric paralysis 312
- Sex as factor in calcareous tendinitis 177
- Shoulder joint anatomy normal, 15 30
- variational, 52 110
- articulations 16
- bursae 25-27
- congenital abnormalities, 45 50
- dislocations 192 245
- acromioclavicular 195 204
- complications 223
- glenohumeral (recurrent) 229-244
- initial, open reduction for 223 224
- McLaughlin's approach 223
- postoperative management 223-224
- luxatio erecta 224
- old, 226-229
- pathology 227
- treatment 227 229
- arthrodesis of glenohumeral joint, 227
- closed methods 227 228
- humeral head resection, 228
- open methods 227 228-229
- untreated, 227
- posterior acute traumatic, 224-226
- roentgenographic examination, 224 225
- recurrent 245
- and injury 245
- pathology 245
- treatment 245
- scapulohumeral, 204 223
- sternoclavicular 192 195
- functional mechanism, 15 30-45
- lesions degenerative 52 110
- ligaments 23-25
- coraco-acromial, 17 18 21 23 25 168
- coracohumeral, 17 23-24 168
- musculotendinous cuff *See* Musculotendinous cuff
- mechanical force requirements 32 38
- Shoulder joint, anatomy normal—(Continued)
- motion at, 30-32
- muscle force couple 30
- movements 28-30
- Shoulder pain of neurogenic origin, 275
- etiology 287
- lesions within spinal cord and canal, 287
- false localization 278
- Spinal muscles paralyzes fascial transplants 410
- Spine cervical. *See* Cervical spine
- Spondylitis ankylosing of cervical spine. *See* Arthritis reumatoid, of cervical spine
- Sprengel's deformity 45-49
- Steindler intra-articular arthrodesis 400-401
- Sternoclavicular joint, anatomy normal, 16 26 27
- dislocation, 192 195
- incidence 192
- treatment, chronic, 192 194
- operative technic, 194-195 196
- Bankart, 196
- Speed, 194-195
- postoperative, 195
- recent cases, 192 193
- motion, 31
- surgical approach, 390
- Sternoclavicular ligament, 27
- reconstruction 195
- Sternocleidomastoid muscle, 32
- Subacromial bursa, 25 27 376
- calcareous deposits mechanism of deposition 180
- pathogenesis 180-183
- acute syndrome 182 183
- chronic syndrome, 183
- clinical features 181
- subacute syndrome, 181 182
- symptoms 181
- symptomatology 180-181
- floor changes in incomplete tears 116
- and glenohumeral joint, 80-81
- pathologic alterations, 180-183
- Subcoracoid pectoralis minor syndrome 305
- Subclavian artery 286 287
- Subclavian vein 286
- Subclavius muscle 36
- Subscapularis tendon calcareous tendinitis in, 180
- Sulcus lesions 100
- Supraclavicular region, anatomy topographic and variational, 283-291
- thoracic operculum, compression syndromes 287
- Suprascapular ligament 17 20
- Suprascapular nerve, 378
- Subscapularis muscle, 19 21 23 24 33 35 36
- Supraspinatus muscle, 17 19 23 24 33 36 38
- function, 114

- Supraspinatus syndrome 112-113
- Supraspinatus tendon 1,
 calcaneous deposits in 138
 calcaneous tendinitis 1, 4, 1, 9, 184
 completely torn and bicipital tenosynovitis 159
 rupture of 41 139
- Surgery 3 2-424
 anatomic considerations 3, 3
 approach. *See* Approach (surgical)
 incisions. *See* Incisions
 indications for and against 3, 2
 landmarks bony 3-5
 of shoulder 3, 4
 position of patient 3, 0
- Synovial capsule 21-23
- Synovial membrane 22 69, 13 5
 hypertrophy 0 2
 lesions musculotendinous cuff 100-10,
 pathology incidence 0, 13
 site 0
- Synoviomelia as cause of shoulder pain 288-289
 clinical features in the upper extremity 289
 involvement of upper spinal segments 289
 pathology 288-289
- Tears of musculotendinous cuff chronic stages
 faulty scapulohumeral rhythm 120
 and good function in supraspinatus region 101-102
 types 102
 history of injury 114
 impaired muscle balance 121
 with strong deltoid, 121
 with weak deltoid, 121
 incomplete 113-114
 bursal side 116
 parallel with cuff fibers 116
 reparative process 116
 within substance of cuff 116
 on synovial side 116
 minor compatible with normal reduction 121
 old 128
 treatment conservative 128
 recent active motions 12
 heat and massage 12,
 relief of pain 12,
 treatment conservative 12, 128
 operative 128
 supraspinatus region 12,
 clinical features 127
 diagnosis 12,
 incomplete 116
 types 116
 treatment surgical acromioplasty 136
 excision of bursa 131
- Tears of musculotendinous cuff—(Continued)
 treatment surgical—(Continued)
 localization of lesion 128
 McLaughlin method 129
- Tendinitis calcaneous. *See* Calcaneous tendinitis
- Tendon of biceps brachii muscle. *See* Biceps tendon
 of pectoralis minor muscle 24
 subscapularis calcaneous tendinitis 1-0
 supraspinatus. *See* Supraspinatus tendon
 Teno-bursite" syndrome 139
- Tenosynovitis adhesive 139
 bicipital. *See* Bicipital tenosynovitis
 of long head of biceps 139
- Teres muscle major 33-38
 minor 19-23, 24, 35-38
- Test Hueter 1, 2
 Ludington 172
 Queckenstedt 288
 Yergason, 1-2
- Thoracic cage evolutionary changes 9
- Torsion angle, in humerus 9-10, 18-19
- Transplantations of muscle 404-409
 biceps brachii 406-409
 deltoid, 408-410
 in deltoid paralysis, 404
 trapezius, 405-406
 triceps brachii 406-409
- Trapezius muscle 32-34, 3,
 paralysis of 42-44
 transplantation of fascial extension 405-40
- Trapezoid ligament 27
- Triceps brachii muscle evolution of 13
 transplantation 406-409
- Tuberculosis of cervical spine, 92
- Tuberosity (ies) and bicipital groove 90-93
 greater fractures. *See* Fractures of greater tuberosity
 lesser fractures. *See* Fractures of lesser tuberosity
- Tumors, of bone. *See* Bone tumors
 of brachial plexus 306
 of cervical spine canal, 28,
 cord 287
 diagnosis 288
 extramedullary 28,
 intramedullary 287
 radicular pain differential diagnosis 28, 288
 of cervical vertebrae multiple 293
 primary 293
 giant-cell. *See* Giant-cell tumor
 intraspinous, cervical spine diagnosis, manometric study 288
 myelography 288
 spinal fluid examination 288
 x ray findings 288

- Scapulothoracic joint, anatomy normal, 16
- Sclerotomes 277
- Scoliosis and arthrodesis of scapulohumeral joint 398
- Segmental pain, deep observation by Inman and Saunders 277
 - false localization 278
- Segmental reference of pain to shoulder and arm
 - causative factors 275
 - neurogenic lesions objective manifestations 275
- Senescence and ruptures of musculotendinous cuff 113
- Serratus anterior muscle 32 35 37
 - paralysis 41-43
 - fascial transplant, 410-411
- Serum neuritis of brachial plexus 308
- Sever's operation for obstetric paralysis 312
- Sex as factor in calcareous tendinitis 17
- Shoulder joint anatomy normal 15-30
 - variational, 32 110
- articulations, 16
- bursae, 25-27
- congenital abnormalities, 43-50
- dislocations, 192 245
 - acromioclavicular 195 204
 - complications 223
 - glenohumeral (recurrent) 229-244
 - initial, open reduction for 223-224
 - McLaughlin's approach, 223
 - postoperative management 223-224
 - luxatio erecta, 224
 - old, 226-229
 - pathology 227
 - treatment, 227 229
 - arthrodesis of glenohumeral joint 227
 - closed methods, 227 228
 - humeral head resection, 228
 - open methods 227 228-229
 - untreated, 227
 - posterior acute traumatic 224-226
 - roentgenographic examination, 224-225
 - recurrent, 245
 - and injury 245
 - pathology 245
 - treatment 245
 - scapulohumeral 204 223
 - sternoclavicular 192 195
- functional mechanism 15 30-45
- lesions degenerative 52 110
- ligaments 23-25
 - coraco-acromial, 17 18 21 23-25 168
 - coracohumeral, 17 23-24 168
 - musculotendinous cuff *See* Musculotendinous cuff
- mechanical force requirements, 32 38
- Shoulder joint anatomy normal—(*Continued*)
 - motion at, 30-32
 - muscle force couple 30
 - movements, 28 30
- Shoulder pain of neurogenic origin 275
 - etiology 287
 - lesions within spinal cord and canal, 287
 - false localization 278
- Spinal muscles, paralysis, fascial transplants 410
- Spine cervical. *See* Cervical spine
- Spondylitis ankylosing of cervical spine. *See* Arthritis, rheumatoid, of cervical spine
- Sprengel's deformity 45-49
- Steindler intra articular arthrodesis 400-401
- Sternoclavicular joint, anatomy normal, 16 26 27
 - dislocation, 192 195
 - incidence 192
 - treatment, chronic 192 194
 - operative technique, 194-195, 196
 - Bankart, 196
 - Speed, 194-195
 - postoperative, 195
 - recent cases, 192 193
 - motion 31
 - surgical approach, 390
- Sternoclavicular ligament, 27
 - reconstruction 195
- Sternocleidomastoid muscle, 32
- Subacromial bursa 25-27, 376
 - calcareous deposits mechanism of deposition 180
 - pathogenesis 180-183
 - acute syndrome 182 183
 - chronic syndrome, 183
 - clinical features 181
 - subacute syndrome, 181 182
 - symptoms 181
 - symptomatology 180-181
 - floor changes in incomplete tears 116
 - and glenohumeral joint 80-81
 - pathologic alterations, 180-183
- Subcoracoid pectoralis minor syndrome, 305
- Subclavian artery 286 287
- Subclavian vein 286
- Subclavius muscle 36
- Subscapularis tendon calcareous tendinitis in, 180
- Sulcus lesions 100
- Supraclavicular region anatomy topographic and variational, 283-291
 - thoracic operculum compression syndromes 287
- Suprascapular ligament, 17 20
- Suprascapular nerve, 378
- Subscapularis muscle 19 21 23 4 33 35 36
- Supraspinatus muscle 17 19 23 24 33 36 38
 - function 114

Tumors of bone—(*Continued*)

- Pancoast, 306 307
- of pulmonary sulcus superior 306
- of spine, cervical. *See* Tumors of cervical spine
- of vertebrae cervical multiple, 293
 - primary 293

Vascular phenomena, with cervical rib 300

Vein(s) 378

- cephalic 375
- subclavian 286

Vertebral column pathologic disorders and associated lesions 291 297

Villi, synovial, 65 69-73 81

Watson Jones extra articular arthrodesis 399-400

Whip-lash mechanism" injury to cervical spine,
293 294

Yergason test, 1, 2

